THE JOURNAL OF THE AMERICAN OSTEOPATHIC ASSOCIATION

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War Service Conferences Coming Soon

PHYSICIANS are under obligation as never before, to themselves and to the public, to assemble and study advances in prevention, diagnosis, and treatment of disease and injury. Doctors' meetings are in the highest degree in the public interest. Their purpose is different from the annual get-togethers and social conventions of many groups having problems which concern only themselves and which could be settled just as well by mail.

Rationing of tires and gasoline has curtailed or eliminated some meetings of small groups where the automobile furnished the chief or only means of transportation. This makes it all the more imperative that there be full attendance at gatherings where for two or three days or more intensive refresher courses are provided, such as at state and provincial or regional osteopathic gatherings which will be frequent within the next few months, and especially at the Annual Conference of the American Osteopathic Association in Kansas City, Mo., running five full days, July 16-20 inclusive, when the best information applicable to present conditions is made available.

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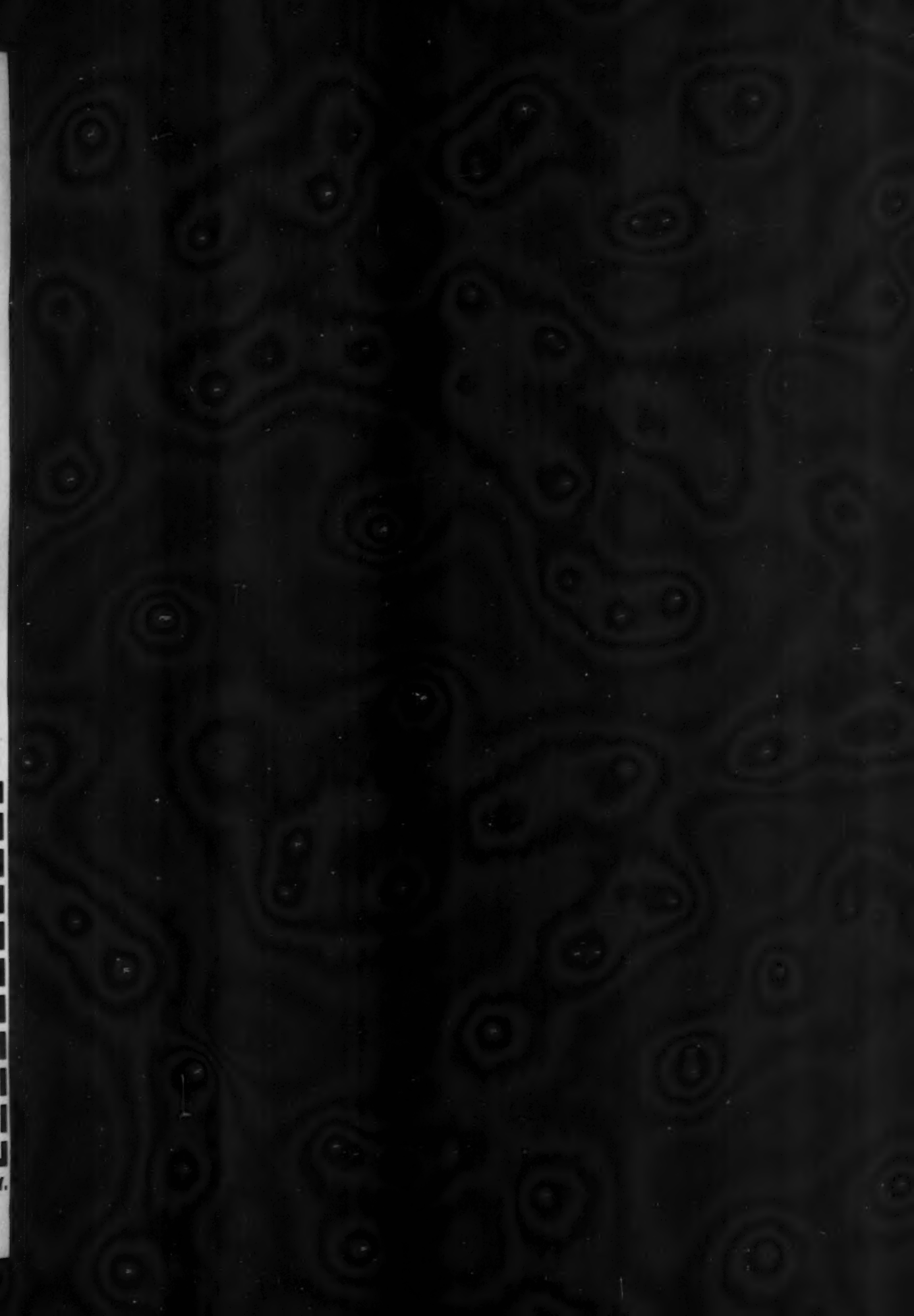

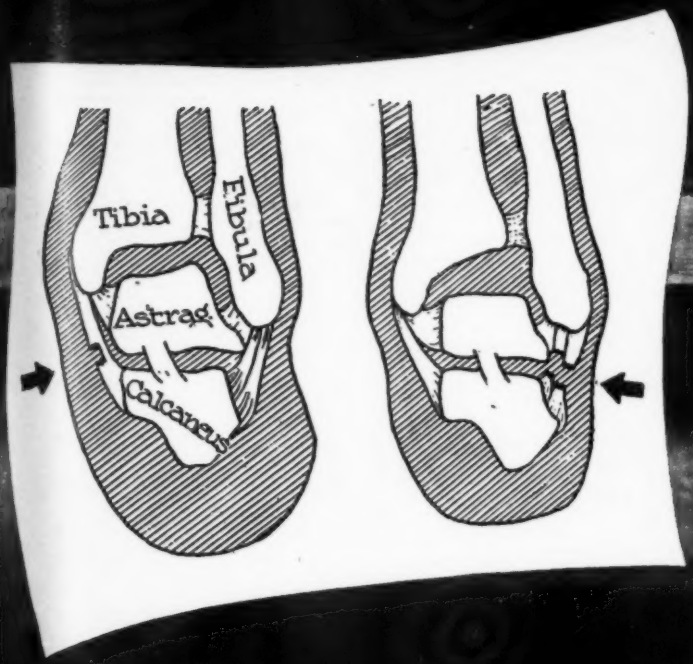

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CONTENTS, JANUARY, 1945

ORIGINAL ARTICLES—

The Applied Anatomy of Some Postural Changes. Angus G. Cathie, D.O., Philadelphia 217

Acute Contagions and Infections in Children. James M. Watson, M.D., D.O., Los Angeles 220

The Problem of Infection in the Chronically Ill Child. Robert S. Roscoe, A.B., D.O., Cleveland 228

Cervical Erosions. M. Carl Frey, D.O., York, Pa. 231

Malaria: Diagnosis and Treatment. Charles C. Tindall, D.O., Kissimmee, Fla. 235

EDITORIALS—

Board of Trustees Meets. Ray G. Hulburt, D.O., Chicago....239

Editorial Policy—A Restatement. Ray G. Hulburt, D.O., Chicago....240

SPECIAL ARTICLE—

The Teaching of Biochemistry. Albert P. Kline, Kirksville, Mo....241

DEPARTMENT OF PROFESSIONAL AFFAIRS—

Committee on Special Membership Effort 244

SURGICAL SUPPLEMENT—

Editorial: Intraperitoneal Sulfonamides. H. E. Donovan, D.O., Raton, N. M...247

A Report of 100 Consecutive Cesarean Sections, 84 Performed under Spinal Anesthesia. Emmett Binkert, D.O., Carson City, Mich....248

A Consideration of Surgery on the Ovary. William T. Barrows, D.O., Oakland, Calif. 251

Surgery of Renal Calculus. W. S. Carey, D.O., Oakland, Calif. 256

The Sterilization of Surgical Rubber Gloves. C. L. Ballinger, D.O., Akron, Ohio 259

DEPARTMENT OF PUBLIC RELATIONS—

OPA Sugar Rationing. C. D. Swope, D.O., Washington, D. C....261

CURRENT MEDICAL LITERATURE....261

CURRENT OSTEOPATHIC LITERATURE 263

STATE BOARDS 264

MEETINGS 264

SUBJECT INDEX

Allergy from synthetic rubber gloves, [Conley] 263-COL

American College of Osteopathic Surgeons:
membership 246-COS
surgical supplement 245-COS

American Osteopathic Association:
board of trustees meets, [Hulburt]....239-E

Anatomy, applied, in postural changes, [Cathie] 217*

Ballinger, C. L.:
Sterilization of surgical rubber gloves....259-COS

Barrows, William T.:
A consideration of surgery on the ovary 251-COS

Becker, Katherine:
Current medical literature....261-COL

Binkert, Emmett:
A report of 100 consecutive cesarean sections, 84 performed under spinal anesthesia 248-COS

Biochemistry, teaching of, [Kline]....241-SA

Blood plasma paragraphs....ad p. 50

Board of trustees of A.O.A. meets, [Hulburt] 239-E

Books Received ....ad p. 37

Carey, W. S.:
Surgery of renal calculus....256-COS

Cathie, Angus G.:
The applied anatomy of some postural changes 217*

Center of gravity, [Hirt, Fries and Hellebrandt] 262-CML

Cervical erosions, [Frey]....231*

Cesarean sections, report of 100, [Binkert] 248-COS

Children:
acute contagions and infections in children 220*
hospital and home care of premature infants: a plan for coordination....ad p. 42
problem of infection in the chronically ill child, [Roscoe]....228*
rural child and the U. S. Children's Bureau, [Lenroot] ....ad p. 38

Convalescent sera in contagious diseases, [Watson] 220*

Convention, Kansas City:
local chairmen 261

Current medical literature....261-CML

Current osteopathic literature....263-COL

Diagnostic errors, common, [Alvarez] 261-CML

Donovan, H. E.:
Intraperitoneal sulfonamides 246-COS

Duffell, R. E.:
Current osteopathic literature....263-COL

Editorial policy—a restatement, [Hulburt] 240-E

Education:
The teaching of biochemistry, [Kline] 241-SA

Essay contest announcement....238

Frey, M. Carl:
Cervical erosions 231*

Gibbs, Stephen B.:
Committee on special membership effort 244-Pr.A

Gynecology:
cervical erosions, [Frey]....231*
consideration of surgery on the ovary, [Barrows] 251-COS

Hospitals:
approved for internship....239-E
hospital and home care of premature infants: a plan for coordination....ad p. 42

Hulburt, Ray G.:
Board of trustees meets....239-E
Editorial policy—a restatement....240-E

Infants: See Children
Infectious and contagious diseases of children, [Watson] 220*
chronically ill child, [Roscoe]....228*

Kidney stone, surgery for, [Carey]....256-COS

Kline, Albert P.:
The teaching of biochemistry....241-SA

Malaria: diagnosis and treatment, [Tindall] 235*

Meetings: See also Convention, Kansas City
announcements 264
official and affiliated organizations....264
special and specialty groups....266

Membership, A.O.A.:
applications ....ad p. 57
changes of address and locations....ad p. 51
Committee on special membership effort, [Gibbs] 244-Pr.A
honor roll 244-Pr.A

Obstetrics:
report of 100 consecutive cesarean sections, 84 performed under spinal anesthesia, [Binkert]....248-COS

Office of Price Administration:
sugar rationing regulations revised..261-Pu.R

Ovary, a consideration of surgery on the [Barrows] 251-COS

Penicillin, pronunciation of....262

Poliomyelitis:
F.D.R. re-dedicates birthday to polio fight, 1945 appeal....ad p. 50

Postural changes, applied anatomy, [Cathie] 217*

Profesional affairs, department of....244-Pr.A

Psychosomatic medicine and tuberculosis, [Weiss] ....ad p. 46

Public relations, department of....261-Pu.R

Renal calculus, surgery of, [Carey]....256-COS

Roscoe, Robert S.:
The problem of infection in the chronically ill child....228*

Rubber gloves, sterilization of, [Ballinger] 259-COS
synthetic rubber glove allergy, [Conley] 263-COL

Russell: son of Phil R. Russell returns from the Philippines, [Hulburt]....239-E

State Boards:
Conn., Ill., Iowa, Mo., N. Y., Ore., R. I., Wash., W. Va., Wyo....264-SB

Sterilization of surgical rubber gloves, [Ballinger] 259-COS

Sulfonamides:
indiscriminate use of, in nasal and sinus infections, [Roberts] 263-COS
intraperitoneal, [Donovan] 246-COS

Surgery: See also American College of Osteopathic Surgeons
on the ovary, [Barows]....251-COS
of renal calculus, [Carey]....256-COS

Syphilis: four-year study emphasizing importance of routine serologic tests for detection, [Dressler and Bruner]..263-COL

Swope, C. D.:
OPA sugar rationing....261-Pu.R

Tindall, Charles C.:
Malaria: diagnosis and treatment....235*

Tuberculosis and psychosomatic medicine, [Weiss] ....ad p. 46

Watson, James M.:
Acute contagions and infections in children 220*

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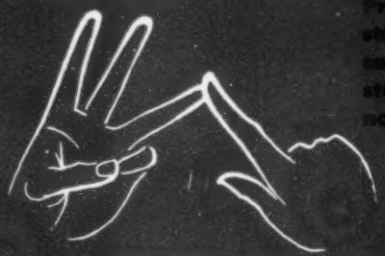

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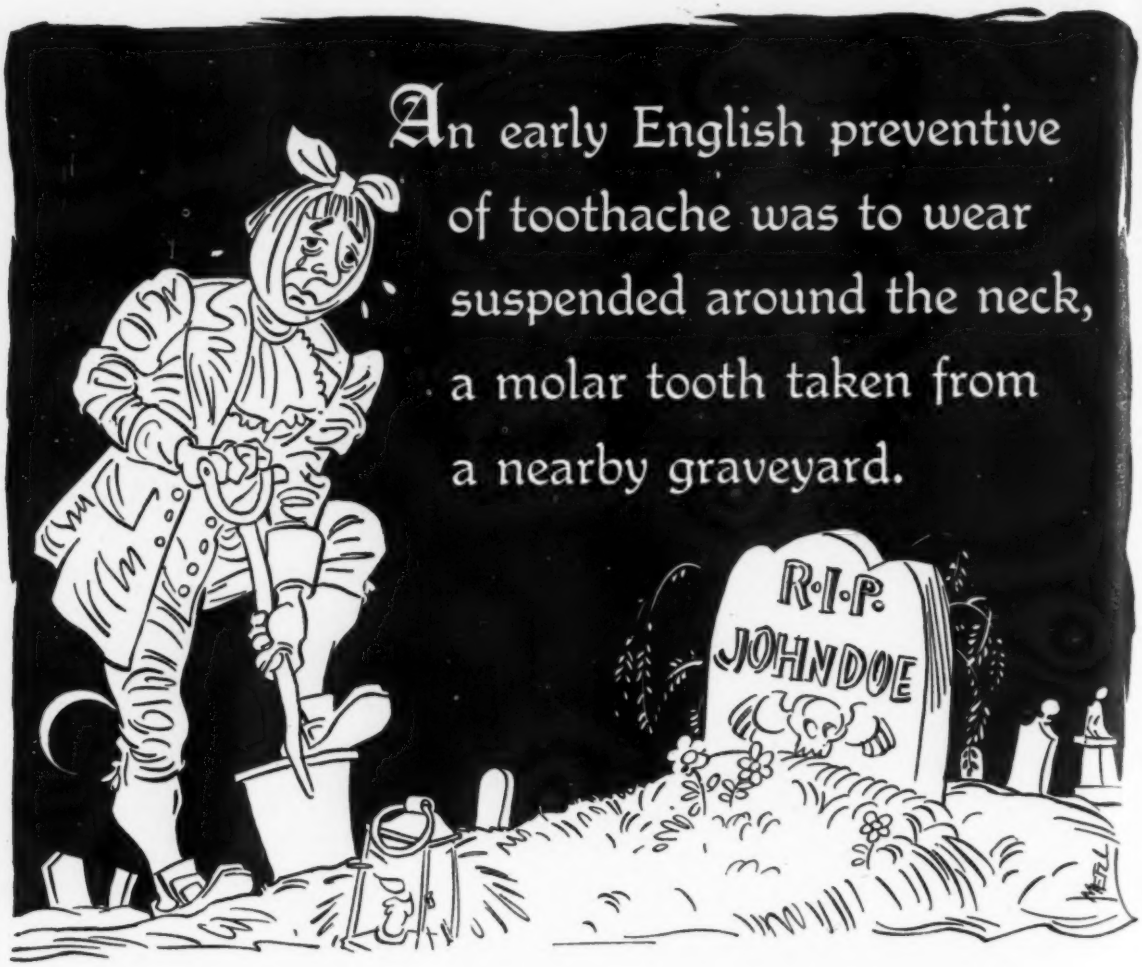
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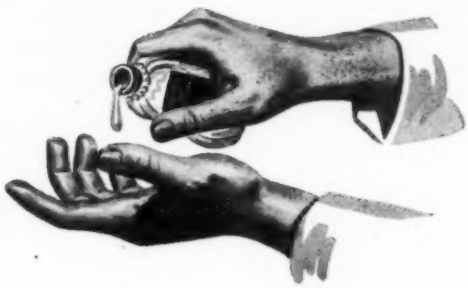

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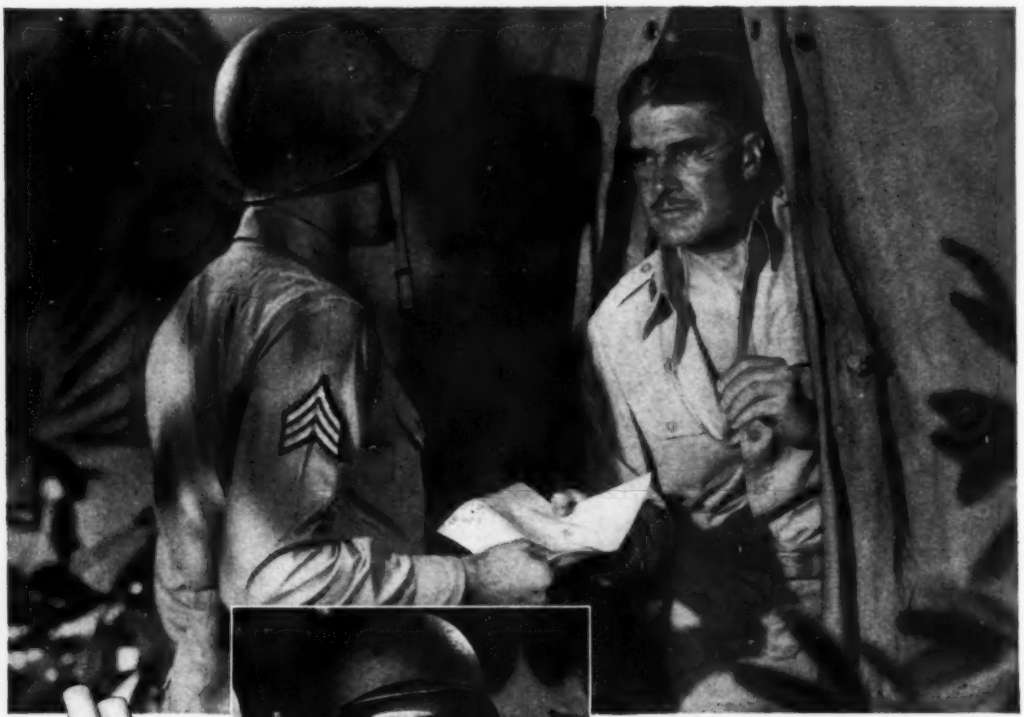

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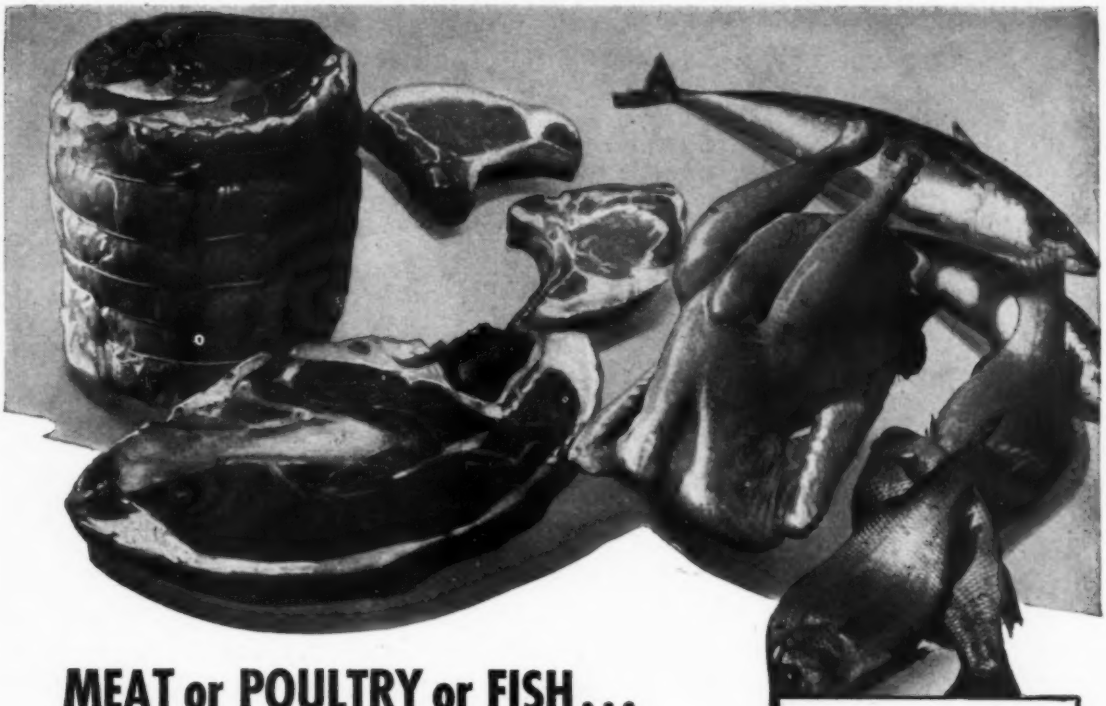

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12% ate none

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In the face of generally poor dietary practices among patients, many doctors recommend a daily supplement of vitamins and minerals. The Vimms formula not only supplies all the vitamins known to be essential in the diet, but unlike most supplements, supplies commonly needed minerals as well—Calcium, Phosphorus and Iron. No product offering its formula in only one tablet or capsule can supply the minerals as well as the vitamins in the Vimms formula.

[1] *Journal of the American Medical Association, February 27, 1943.*

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600 USP Units	C	600 USP Units
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10 mg.	Niacin	10 mg.

In addition, Vimms supply the minerals most frequently deficient in the average diet

CALCIUM	375 mg.
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*Journal of the A.M.A., July 18, 1942, pp. 948-9

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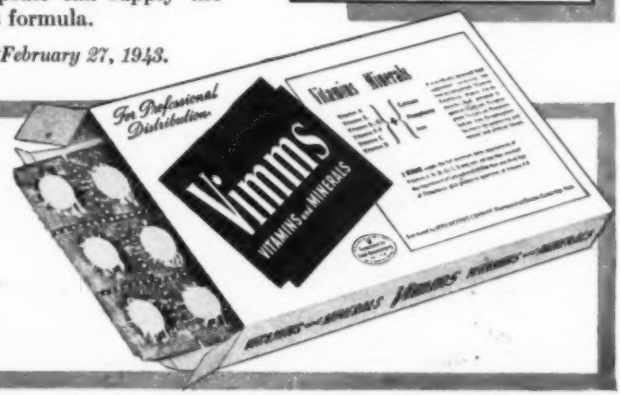

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*Burke, B. S., Harding, V. V. and Stuart, H. C.: Nutrition Studies During Pregnancy, JI. Ped. 23; 506-515 (Nov.) 1943.

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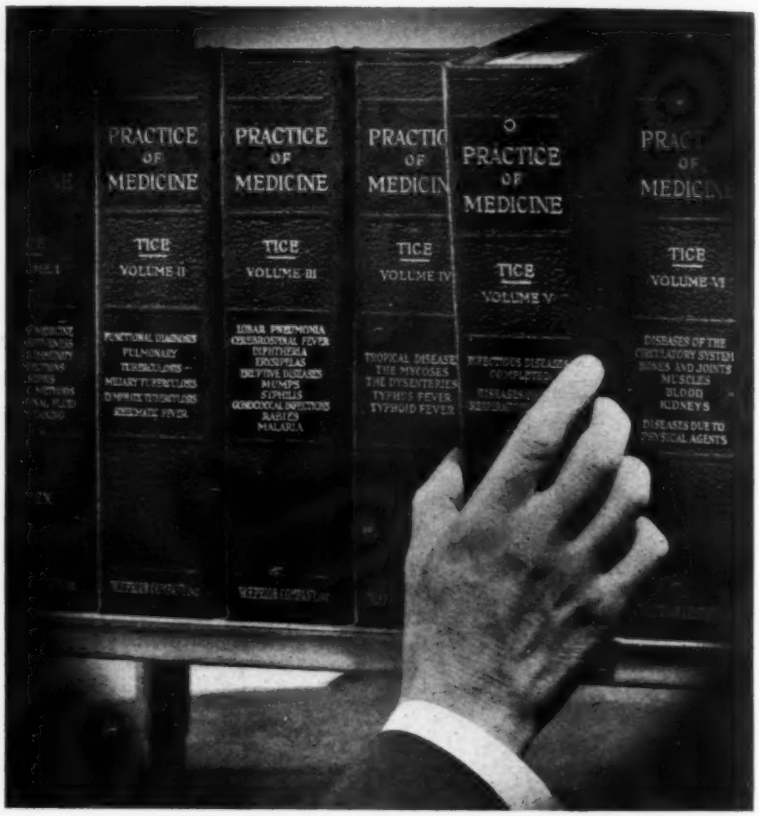

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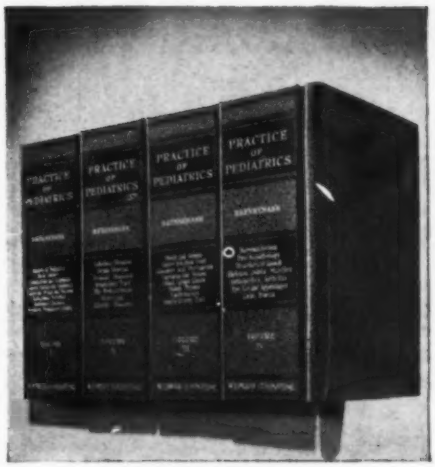

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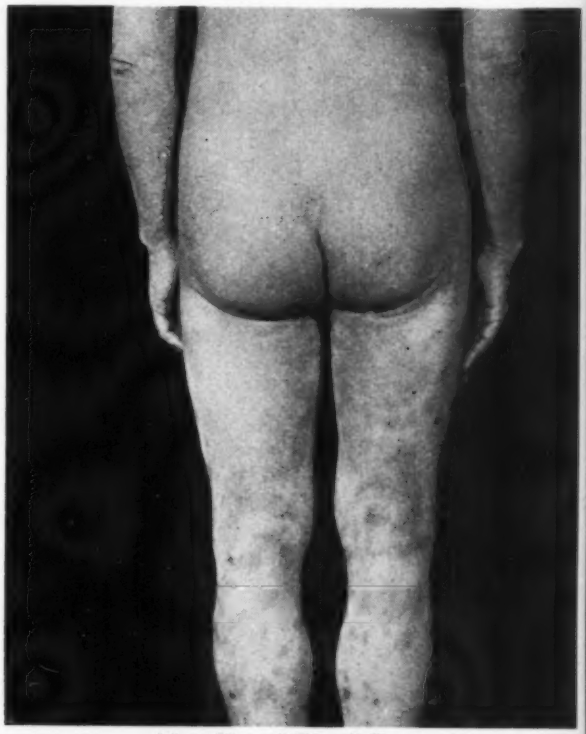

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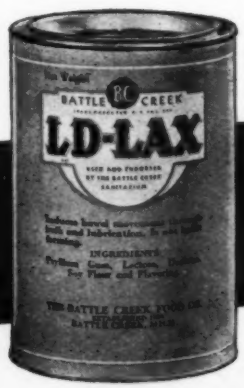

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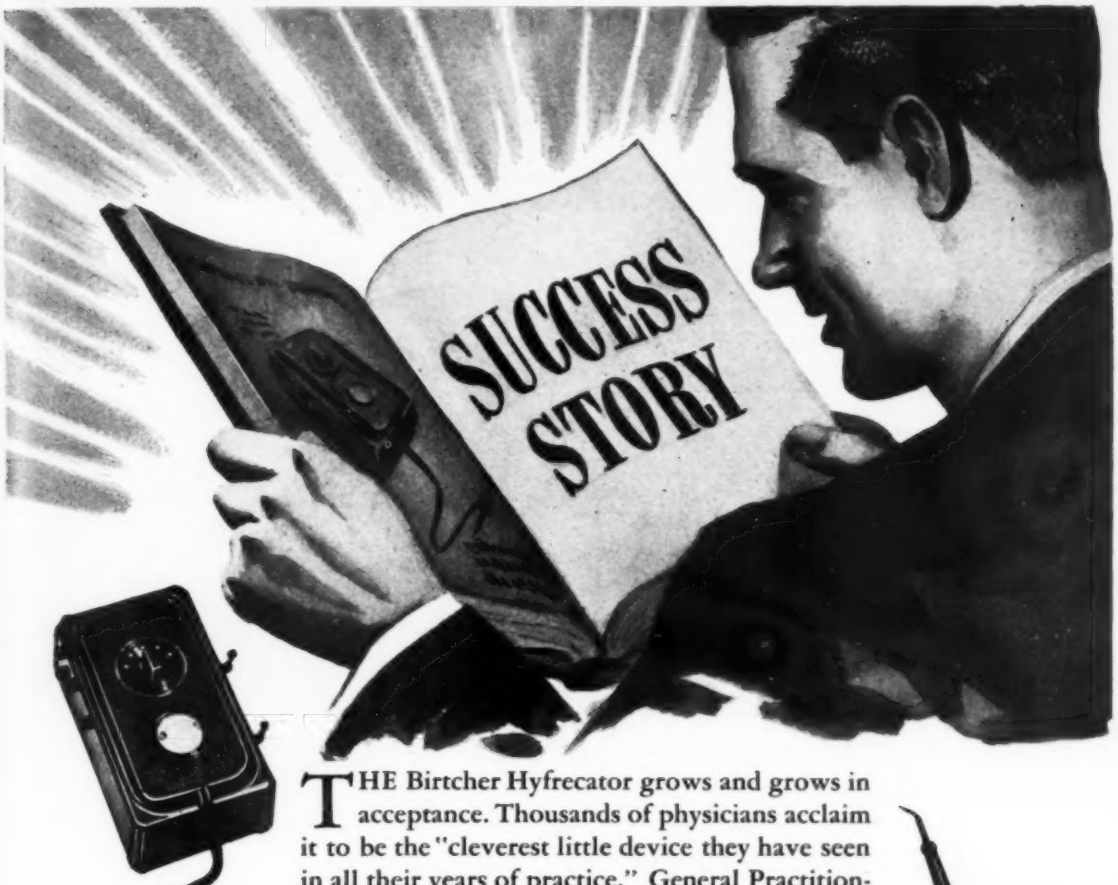

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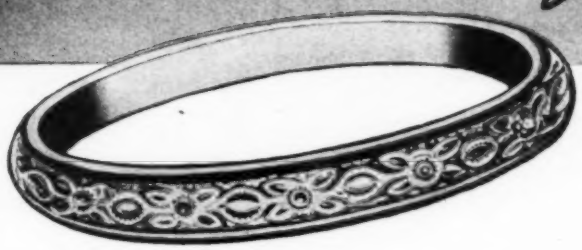

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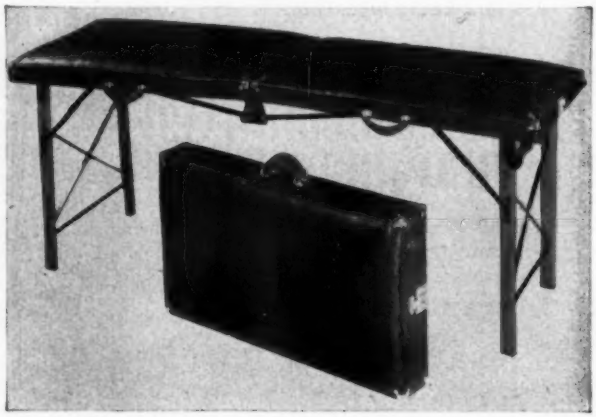

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The Applied Anatomy of Some Postural Changes*

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Department of Anatomy

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Philadelphia

The erect position of man is the result of a series of evolutionary changes. The student of comparative anatomy recognizes similar structures in various lower forms of life; he finds special adaptations of pre-existing parts to serve the requirements of each more advanced form. He finds the predecessors of man gradually assuming the upright position and depending less and less upon their forelimbs for purposes of locomotion. He notes that the center of gravity changes. So similar are man's general structural characters to those of his closer relatives in the zoological classification that they serve a very useful purpose in assisting us in the understanding of mechanical technicalities and postural problems. Nature has not been prodigal in the production of new parts for the architecture of Homo sapiens, but, instead, has taken preexisting parts and, after making minor changes, has assigned them new and more complicated duties and permitted him to assume the upright position

A knowledge of these facts is valuable to the physician if he is to appreciate the liabilities accepted by man as he assumed this position. He will examine and treat man's postural defects more intelligently if he recalls that the more recently acquired actions of muscles and motions of joints are often the first to be lost and the last to be regained. Mackenzie[1] says, "The particular functions of a muscle are the result of the evolutionary history of that muscle." Thus, in the treatment and re-education of parts, the simpler motions must be perfected before the more complicated can be regained.

At the time of birth the infant is unable to assume the upright position. He spends the greater part of three years before he acquires the art of balance. During this period, he is seen to reproduce gradually the posture of his nearest relatives in the zoological classification—the anthropoid apes. During this experimental period, his skeletal, muscular, and nervous systems are especially vulnerable to attack.

Having assumed the upright position, individuals are classified as having one of three types of bodily habitus: longilineal, brevilineal or intermediate. Each type indicates certain constitutional tendencies. The longilineal type is frequently the victim of an irritable nervous system, acute infections, respiratory diseases, gastrointestinal disturbances, low-lying viscera, and marked postural defects. The brevilineal type is more likely to suffer from high blood pressure, cardiorenal diseases, bronchitis, asthma, and pneumonia. Because of his substantial build, he is less liable to present postural problems than the preceding type. The intermediate type is well-balanced physically and mentally. Regardless of the variations in bodily types, general anatomical relations should remain unaltered and, under normal conditions, the principles of equilibrium and balance are the same.

The center of gravity of the body is in the upper lumbar region.[2] A line projected vertically upward from this point would pass through the atlanto-occipital or craniospinal junction, while one projected vertically downward from the same point would pass through a line connecting the talonavicular joints. The sacroiliac, hip, knee, and ankle joints all lie posterior to this line.

Some may consider the construction of such a line a debatable point, calling attention to the large number of postural variations seen every day among persons who present no symptoms suggestive of mechanical dysfunction. These are admitted, but it must be realized that the appropriate compensatory changes have been made. These persons represent potential postural problems when their structural compensation is broken down.

"From a mechanical point of view, the ideal posture is one in which the different segments of the body, head, neck, chest and abdomen are balanced vertically one upon the other so that the weight is borne mainly by the bony framework, with a minimum of effort and strain on muscles and ligaments; this is the long axis of the segment, seen in profile, from a vertical line instead of zigzag."[3]

For such a posture to be possible, normal postural reflexes must be in operation, maintaining perfect balance between opposing muscle groups. MacGregor[4] calls attention to the postural activity of certain mus-

*Delivered before the Annual Meeting of the Missouri Osteopathic Association, St. Louis, October 18, 1944.

cles. Of these he says: "Muscles concerned with keeping the trunk erect have a special physiological property called 'Postural Activity'." This implies that their activity is constantly greater than that of other muscles which do not participate in this activity. This group of muscles includes the sacrospinalis, glutei, quadriceps, sartorius, and to a certain degree, the muscles of the calf of the leg. It has been suggested that a special center in the cord controls this activity.

The skeleton of Homo sapiens is characterized by appendages adapted for the performance of special tasks. The upper extremities are highly specialized appendages capable of complicated movements demanding remarkable coordination of muscle groups. Their bones, relieved of most of their weight-bearing, are smaller and the joints exhibit a greater range of motion than those of the lower appendages. The lower extremities are designed for weight-bearing and locomotion. Their bones are larger, the joints stronger, and their range of motion more restricted than those of the upper extremities. The muscles of the lower extremities are larger, coarser, and less highly specialized than those of the upper. The hip joint is placed in more intimate relation with the spine than is the shoulder.

The weight of the body segments is continually exerting a downward force upon the more caudally placed units. From the upper spinal regions, the weight is transmitted through the sacrum to the femorosacral arch. Examination of the pelvis reveals that the transmission of the weight is through a strong and thickened portion of the innominate bone, to the upper part of the acetabulum. Weight transmission in the presence of correct posture is not attended by joint strain. The bones, ligaments and muscle groups are in perfect balance. Disturbances of balance from any cause disturb weight bearing and produce joint strain and, eventually, deformity.

Since the causes of bad posture are numerous, only the more important ones will be mentioned. Malalignment of the skeleton as the result of trauma is frequently accompanied by disturbances of balance. Until this is corrected, postural defects may occur. Diseases of bones and joints accompanied by productive or destructive changes are potent enemies of good posture. Closely associated with bone disease is disease of the muscular system because the latter is accompanied by changes in joint tension and position. Partial or complete loss of action in one group of muscles permits their natural antagonists to act without opposition. Limitation of motion and changes in the relations of articular surfaces result. Diseases of the central and peripheral nervous systems produce their effects by disturbing normal muscle action, joint stability and body balance. Nutritional disturbances resulting in impaired trophicity, fatigue, and the general weakness which so frequently accompanies convalescence, are responsible for a large number of postural defects.

Habit and occupation are both associated with position of parts in a certain attitude over a protracted period. The normal segmental weight-bearing of the body is disturbed and that function is often transferred to a part not intended for it. The importance of these two common causes should not be underestimated. They often become effective early in life when the developing framework of the child is subjected to sitting in a schoolroom.

Another, but perhaps less common, cause is that group of conditions which produce asymmetry because of a unilateral reduction in the size, or depletion, of a part of the body as, for example, atelectasis, massive fibrosis of one lung, and amputation of an upper or lower extremity. These conditions are especially active in the production of scoliotic changes. The strong tendency of nature to keep the eyes on a horizontal plane accounts for the scoliosis that so characteristically accompanies torticollis. Instances in which poor posture has been assumed for the relief of pain are well known. They should be carefully studied since much may be learned concerning the origin of that pain.

The various systems of the body are interrelated in so many ways that in considering changes in one the others must be noted if our evaluations are to be correct. Correct posture depends upon a skeleton composed of a group of properly constructed parts, held in correct relation, and controlled by muscles acting under the direct influence of the nervous system. For the proper nutrition of these systems, normal metabolic processes are essential. When poor posture exists, unfavorable changes take place, altering the structure of parts, disturbing their normal relations, and normal muscular balance is lost. Various symptoms referable to the central or the peripheral nervous system, or both, and to vascular structures, with or without impairment of metabolic processes, are usually coexistent in some degree.

Time will permit mentioning only the more important structural changes resulting from poor posture. Excluding those following severe trauma, the majority of marked postural deformities develop slowly. They are the result of a series of changes. From the onset, there is some disturbance of vertebral mechanics since the weight-bearing is shifted, producing varying degrees of joint strain. Ligamentous and muscular shortening on one side with lengthening on the other is a common finding. Articular surfaces so held undergo alterations in size and direction. Productive bone changes involving the articular processes are frequent. There is radiographic evidence to show joint change, thickening of the articular capsule, marginal spondylitis of the vertebral bodies, and increased muscular tension.

Many of the subjects studied in the anatomy laboratory of the Philadelphia College of Osteopathy show asymmetry in the depth of the sides of the vertebral bodies with peripheral lipping or ankylosis, although the majority have a fair amount of preservation of disc spacing. It is interesting and highly significant to note that productive bone changes projecting from the posterior margin of the periphery of the bodies are seldom found invading the vertebral canal. A previously thickened ligamentum flavum, now partially ossified, is sometimes demonstrated projecting from the lamina. It is capable of producing root pain—radiculitis. Extensive ossification of the anterior longitudinal ligament is also found in some of these subjects. Among the common changes found in the vertebral arches are the alterations of facet facing and size, increase in the bulk and density of articular processes, depressions at the base of superior articular facets. The last mentioned changes are the result of pressure from the lower margin of the inferior articular process of the segment above. Increase in the length and obliquity of the spinous processes with

extensive bridging is often found in the thoracic region. In the lumbar region abnormal "facets" are often found on the upper and lower margins of the spinous processes, ankylosis being complete in some. Many of these changes are the result of a shift of weight-bearing to structures not originally intended for its reception. Increase over the normal forward tilt of the pelvis is seen in many. In some of the more marked cases it is accompanied by distortion of the acetabula and femoral heads. Further investigation of these changes is being made at this time. Studies of articular facets are being carried out by the Departments of Radiology and of Anatomy, participating with the Research Department of the Philadelphia College of Osteopathy.

In a general way, "poor posture" refers to the stoop-shouldered, flat-chested individual with a "hollow" back and a pelvis tilted well forward. Altered tension of muscles and ligaments, accompanied by joint strain and improper weight-bearing is widespread. Although stooped shoulders are said to be the commonest of postural defects, they are usually associated with, or the precursor of, other deformities.

When one considers the number of occupations that require the head to be bent forward and the arms carried in front of the body, it is not surprising that this condition frequently exists. The student in school, the person sitting behind a desk or bench during the pursuit of his occupation, the housewife sewing, are all spending hours in this postural attitude. It is an example of the habitual use of parts in a certain position which affects the length of muscles and ligaments, alters the weight-bearing surfaces, disturbs the center of gravity, and leads to deformity.

The stoop-shouldered individual is seen to carry the head forward. The cervical curve is decreased and the thoracic curve increased by flexion. The shoulders are drawn forward and the chest is flattened. There is usually abdominal prominence below the level of the unbilicus.

In such cases, it is necessary to consider a succession of abnormal relaxation in some muscles, followed by shortening, stretching, and loss of tone in others associated with joint strain. As the head is brought forward by the flexing cervical segments, the scaleni muscles are permitted to relax, thus lessening the support of the upper ribs. The tendency is for the chest wall to become flattened anteriorly. The cervical flexion is followed by an increase of the thoracic curvature, and in both regions the tension of the spinal musculature increases. The scapulae become abducted, and the weight of the shoulder girdle and upper extremity further increase and maintain the spinal deformity. These altered relations increase the distance between the origin and insertion of the trapezius, of the rhomboidei major and minor, and of the levator scapulae which result in strain, stretch, and eventual loss of tone. The abduction of the scapulae causes a lowering of the coracoid process which brings the origin and insertion of the pectoralis minor closer together, further depriving the anterior chest wall of support, and favoring flattening.

The tips of the shoulders have now assumed a position that is downward and forward, bringing the origin and insertion of the serratus anterior, and of the pectoralis major and minor muscles closer together. After a period of relaxation, their chronic shortening takes place.

The exaggeration of the lumbar curve is accompanied by a shift of the weight to the posterior part of the vertebral bodies and to the articular processes. The weight is delivered to the pelvis through the lumbosacral arthrodia, producing maximum joint strain of this transitional area. Radiographic examination and study of laboratory specimens show most marked changes of and around this joint. The increased forward inclination of the pelvis and instability of the hip joint are next in the order of changes. These favor the shortening of the erector spinae group and flexors of the hips accompanied by lengthening of the abdominal and hamstring muscles. Such muscular imbalance serves to maintain the deformity.

The musclar changes mentioned are by no means the complete picture. Due to their action, the joints throughout the involved regions are subjected to continual strain. Inflammatory processes followed by articular and periarticular changes appear. The relation of the articular processes to the intervertebral canal containing the funicular portion of the spinal nerve is important. Change here sometimes causes a reduction in the size, and alteration in the shape, of the canal. Alteration in the position of the ribs is accompanied by changes in the costotransverse and costovertebral joints. This is of special significance when we recall the intimate relation that exists between the costovertebral joints and the lateral chain ganglia of the sympathetic division of the autonomic nervous system. The possibility of irritation to the spinal nerves and autonomic imbalance has now been added.

The flattened chest wall restricts the normal respiratory movements. This impairs respiratory function, predisposing the individual to malnutrition and pulmonary infections.

Although the changes which take place below the level of the diaphragm are sufficient in themselves for a complete study, a few of them should be called briefly to mind. Lowering of the costal arch accompanies the flattening of the chest wall already described. The origins and insertions of the muscles of the anterior and anterolateral abdominal wall are brought closer together by this change, permitting prominence of this region below the level of the umbilicus. The distance between the origin and insertion of the diaphragm is decreased by the reduced size of the thoracic outlet. The diaphragm is lowered, causing increased tension on the pericardium with its neurovascular contents. The support given to the abdominal viscera is decreased. This results in disturbances of visceral position and function. These changes are often associated with mesenteric drag and an increase in pressure being exerted upon the pelvic viscera. The lack of adequate muscular tension and the associated mesenteric drag lead to dilatation of the intra-abdominal vessels further favoring visceral disease.

It is not my purpose to outline the detailed treatment of these cases but to call attention to certain general principles regarding their management. The purpose of the treatment is to lengthen the shortened muscles and to increase the tone of those in which it is diminished or lost. It is important to remember that *too sudden or too forceful movements will only superimpose trauma on already damaged tissues.* The type of treatment given and exercise outlined must be governed by the age and resistance of the patient, the

degree of muscular impairment, and the extent of articular and periarticular changes.

It is taken for granted that such manipulative treatment as may be indicated will be administered in all cases. In early cases showing minimal changes, the patient should also be instructed in the use of exercises devised for the prevention of deformity. In the more advanced cases, the muscles being far from normal cannot be expected to respond well to exercises devised for normal ones. In these cases, the exercises outlined should require only a small number of their normal capacity of work units. Such reduction in the number of work units may call for exercises carried out in positions of rest. It is here that the ancestral history of muscles will be of value in planning their re-educational program and the principles of muscle action will serve the physician and his patient best.

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Acute Contagions and Infections in Children*

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The purpose of this paper is to review the subject of infectious and contagious diseases in infants and children. Because of the scope of this subject, emphasis will be laid on the newer developments only. Time will not permit me to mention all the diseases, but some will be selected which have been discussed in recent literature and which have been dealt with in the writer's experience. These include: Malaria, epidemic diarrhea of the newborn, influenzal, pneumococcus and streptococcus meningitides, measles, scarlet fever, staphylococcus sepsis, tetanus, coccidioidosis, and poliomyelitis. Therapeutic measures such as the sulfa drugs, penicillin, convalescent human sera and x-ray therapy also will be considered.

MALARIA

Prominent in the literature because of the experience of the armed forces, malaria is being brought more and more to our attention. Malaria is very prevalent in many areas of the United States, and there the practicing physicians are on the alert for it, and their experience leads them to recognize its protean manifestations. But in communities like Los Angeles and Southern California it is, I think, often overlooked. Every now and then I run across a case of malaria that apparently has originated in Los Angeles County and has not been imported from some typical malaria area.

When malaria occurs in infants neurological symptoms are likely to be prominent, resembling meningitis and encephalitis. The two following brief case reports will illustrate this point.

One case that I encountered was in a newborn twin that I delivered. The other twin was stillborn and macerated. The live infant seemed quite normal at birth, but about the third day after birth developed attacks of cyanosis with convulsions. Between attacks the infant was in a state of hypertonicity with extension of the extremities to the degree of opisthotonos. The temperature was markedly elevated. Thinking the infant had meningitis, I did a spinal tap; but the spinal fluid was normal. Blood smears taken on the next day in the course of a routine blood count showed the malarial organism, and the infant recovered under quinine therapy. The above case occurred some twenty years ago, and is recounted from memory.

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I saw the second case in July, 1943. An infant, aged six months, was taken to the Children's Hospital with a tentative diagnosis of encephalitis. He had had three attacks of convulsions at monthly intervals preceding his admittance to the hospital. During his stay he was in an oxygen tent and cyanotic much of the time, with repeated convulsions. A staphylococcus was cultured from the blood. This may have been due to laboratory contamination. The working diagnosis was encephalitis, but spinal fluid studies revealed normal findings and no virus was isolated. At the time of discharge on August 9, the diagnosis was "probable encephalitis, possible epilepsy." He was apparently both blind and deaf, and had a very large abdomen. After returning home, he contracted whooping cough. In November, at the age of thirteen months, he was unable to sit or speak. He had periods of great irritability; but neurological examination revealed nothing abnormal except that he seemed to be greatly retarded.

At the age of sixteen months he was brought to me for treatment for "Jacksonian epilepsy." On examination I found him to be well nourished but pale. He was being treated by one of the osteopathic physicians who had given him large doses of vitamin B, iron, ultraviolet irradiations, and osteopathic manipulative treatment. His blood count showed a hemoglobin of 50 per cent, a red cell count of 3,030,000, a white cell count of between 4,000 and 5,000, and a differential count which was normal except that the Schilling shift was to the right. He could not speak and he could not stand. He now had hyperactive knee jerks. There was enlargement of the spleen halfway to the umbilicus; the liver was not enlarged. Because of the history and enlarged spleen, I suspected malaria. After one-half cc. adrenalin was administered, thick blood smears were taken, and quartan malarial parasites were found. Atabrine and quinine therapy has brought about marked clinical improvement with cessation of the convulsions. The infant is, however, still mentally retarded.

The first case mentioned illustrates the tendency of infants to have attacks of cyanosis and convulsions as the equivalent of chills, without the presence of an encephalitis or meningitis. The second, no doubt, represents a true case of malarial encephalitis.

Other aspects of the malaria problem that should not be lost sight of are: (1) It can give false positive serological reactions;[1] (2) parasites may be found in the blood of patients that do not give a history of being from a malarial district or of ever having had malaria; (3) malaria may be contracted with no symptoms of active disease until months after infection, particularly when antimalarial drugs are taken prophylactically.[2]

EPIDEMIC DIARRHEA

Epidemic diarrhea in the newborn has often been the cause of high mortality in hospital nurseries There is some confusion in the literature as to its classification from the standpoint of cause. Classical epidemic diarrhea of the newborn, with numerous loose, watery, greenish, gassy, squirty stools, may well be due to the virus of the common cold or the influenza virus, as repeated bacteriological studies of the stools in such epidemics have shown no bacterial cause. However, in other epidemics organisms of the *Salmonella* and dysentery groups have been found, and in other instances parenteral infections have been incriminated.

In *Medical Clinics of North America*, May, 1939, Abramson, Frant, and Oldenbusch[3] draw a distinction between epidemic diarrhea and *Salmonella* infections of the newborn. They tabulate the differential diagnostic features, of which the following are the most important:

Epidemic diarrhea is specifically limited to the newborn, with an incubation period of from two to twenty days, and an average of seven. The onset is gradual, referable to the gastrointestinal tract; the invasion is gradual with symptoms of gastroenteritis; there is little or no fever sometimes for two or three days, and the fever results from dehydration rather than toxemia. There are no convulsions. Later intense alimentary intoxication develops with temperatures from 100 to 104 F. There is marked dehydration and acidosis. Stools are yellow and watery with no blood or pus, and there are symptoms of marked acidosis. On physical examination there is no sign of parenteral infection, but there is marked dehydration, toxicity, and shock. However, there may be terminal otitis media and bronchial pneumonia. Pathology is mainly the consequence of severe dehydration, and the infectious agent is unknown.

In contrast, in *Salmonella* infection of the newborn the incubation period is shorter, from three to four days. The onset is sudden with intestinal symptoms, but often with respiratory and central nervous system symptoms as well. The temperature is higher; convulsions are frequent; there are rice-water stools; bacteremia may be demonstrated; the course may be very brief or of a prolonged typhoidal type. On physical examination, besides the signs of gastrointestinal involvement, as a result of bacteremia there may be involvement of the central nervous system (meningitis), lungs (pneumonia), and skeletal system; and rose spots and purpura may occur. The *Salmonella* organism may be recovered from stools, blood, urine, and exudate; and there will be positive blood agglutination tests. Bacillary dysentery must also be differentiated from these two conditions.

In epidemic diarrhea, pathologically the extreme and rapid dehydration with its concomitant salt balance disturbance seems to be as largely responsible for the deaths as does the toxemia of the infection. Extreme blood concentration may result in sinus thrombosis, and cause the death of a patient who otherwise might have recovered.

In the last two years in Los Angeles, several of the M.D. hospital nurseries were visited by such epidemics with mortality rates reported to be as high as fifty per cent. An epidemic in Monte Sano Hospital nursery three years ago affected about one-third of the babies, and the mortality rate was zero. In the Los Angeles County Osteopathic Hospital, we have occasion to handle these cases frequently. They are usually brought in from the outside.

Based upon present conceptions of its cause, epidemiology, and pathology, our treatment is as follows:†

1. Complete stool examinations and cultures for evidence of bacterial, amebic or protozoan infestation.

2. Rest of the intestinal tract for twelve to twenty-four hours—nothing administered by mouth except water.

3. In the meantime administration of fluids by hypodermoclysis or intravenous drip, thus restoring fluid and mineral balance.

4. After dehydration is overcome, the administration of sulfathiazole or sulfadiazine. (Where enteral infection is demonstrated or suspected, sulfaguanadine or sulfasuxidine is used. The successful treatment of epidemic diarrhea of the newborn with succinylsulfathiazole is reported by Twyman and Horton[4] in the *Journal of the American Medical Association* for September 18, 1943. The dosage was two grains for each pound of body weight, followed by one-sixth the initial dose every four hours. They administered vitamin K because the drug reduced the number of coliform organisms, with the result that the absorption of the vitamin was reduced. Vitamin K can be given hypodermically.)

5. After the 12 to 24 hour fast, the feeding of protein milk or skimmed boiled milk to which Casec is added, or a formula may be composed of Dryco with the addition of Casec. (In any case the principle of high protein feeding is followed. Buttermilk or acidified protein milk may often be used satisfactorily in either putrefactive or fermentative diarrheas. The amount of food and frequency of feedings should be determined by the infant's appetite. Where feeding has to be routinized, the four-hour interval is probably best. In cases of diarrhea with foul-smelling alkaline stools, the pectin agar diet may be used for a time, gradually changing to a normal milk formula.)

6. After dehydration is overcome, transfusions of whole blood, from 50 to 100 cc. repeated every day or every second day for two or three times. (This gives great support to the immune processes and added nutrition to the infant. Plasma may be used where whole blood is not available. Specific antiserum treatment is used when a bacteriological diagnosis is made and a serum is available. Paregoric is used for extreme restlessness and pain. This is seldom necessary, but it is very helpful when it is needed.)

7. The observance of complete aseptic technic.

Much might be said in elucidation of what is adequate technic in the handling of contagious diseases,

†Glaser, K., and Bruce, J. W.: Treatment of epidemic diarrheas and dysenteries in infants and young children; comparative study of different treatments and their results. J. Pediat. 24:53-61, Jan. 1944.

Lembcke, P. A.: Quinlivan, J. J., and Orchard, N. G.: Epidemic diarrhea of the newborn; report of two outbreaks. Am. J. Pub. Health 33:1263-1273, Oct. 1943.

House Staff Manual, Los Angeles County Hospital.

also as to the technic of intravenous work in the newborn and small infant, but space will not permit. The dosage of sulfonamides need not be repeated here.

MENINGITIDES

Many importantant advances have been made in the treatment of the meningitides in recent years. Specific sera, the sulfonamides, and penicillin have been applied more efficiently and successfully. A great advance in the treatment of epidemic meningitis was made with the advent of antimeningococcic serum. This was used at first intrathecally, and the mortality rate was reduced from between 70 and 80 to between 30 and 50 per cent. But patients with fulminating meningococcemia usually died. These were the cases that either because of an unusually virulent strain or because of very poor resistance, could not meet the first onslaughts of the disease; or because of hemorrhage in the suprarenals there developed a Waterhouse-Friderichsen syndrome with multiple hemorrhages, cyanosis, hyperpyrexia, and then collapse and death. I have observed the syndrome more often in infants under the age of two. I have observed the so-called fleabite rash of meningococcemia also more often in infants.

Even yet we do not have adequate measures to meet this emergency. I can remember having read one report wherein this condition was met successfully by enormous doses of adrenal cortex hormone. The onset in the infant of meningococcic meningitis is not always so abrupt. Sometimes it is very insidious and the symptoms referring to the nervous system are at a minimum.

I remember one case in an infant about three months of age which was brought into the Los Angeles County Osteopathic Hospital with a complaint of vomiting. Physical examination showed an infant in a fairly good state of nutrition, mildly dehydrated because of the persistent forceful nature of the vomiting. The intern suspected pyloric stenosis, and instituted a gastrointestinal x-ray series which did not reveal any abnormality. The temperature never went over 101.6 F. by rectum, and the blood count showed mild leukocytosis with increases in the polymorphonuclear count. I was called in to see the baby on the supposition that it was some gastrointestinal upset. I found it mildly hypertonic, with more extension than flexion of the extremities, slightly resistant to forward bending of the neck, with hyperactive reflexes and a slightly full fontanel. I ordered a spinal tap, and we found purulent fluid with many meningococci. The infant had by now been sick over two weeks. We instituted treatment with serum intraspinally and antitoxin intramuscularly, and the patient made a complete recovery.

Years ago I treated a boy successfully in the home, for epidemic meningitis using the serum intraspinally. One month later, his sister, who was about seven years of age, developed a fulminating case with convulsions, delirium, skin rash, very high fever, and a piercing cephalic cry. She vomited large quantities of round worms and also passed large quantities by rectum. Serum treatment was of no avail. This, I think, illlustrates the importances of the constitutional state on the recovery from the disease. Subsequent examination of her brother did not reveal worms.

Carriers, who spread the infection during epidemics in spite of quarantine, can be rendered free from meningococci in four days by treatment with sulfadiazine, and it could be used as a prophylactic measure in members of a family or with other known contacts of meningitis cases.[5]

The introduction of antimeningococcic antitoxin seems to have been an advance in the treatment of the disease, and figures indicate the drop in mortality to between 15 and 30 per cent. The technic of giving the antitoxin is easier than formerly in that it does not require intrathecal administration. It is given intramuscularly and intravenously. Spinal taps are done for diagnostic, symptomatic, and follow-up purposes. My personal experience has led me to believe that the intrathecal administration of the serum could often be used to advantage in cases where the antitoxin was also used, the serum aiding in stimulating phagocytosis. Serum should be given with human serum for its complement content.

With the advent of the sulfonamides, another great advance occurred in the treatment of meningococcus meningitis and meningococcus sepsis. Mortality rates dropped as low as 10 per cent, even in epidemics. Sulfanilamide was at first used. It could be given in eight-tenths of one per cent, subcutaneously or intravenously, and could be given intraspinally as neoprontosil, 5 per cent, 2 to 10 cc. Of course its toxic effects had to be prevented and treated. Each new sulfa drug has reduced these toxic risks and effects without seemingly impairing the antimeningococcic efficiency. The drug can be given intravenously as the soluble sodium salts, thus meeting very efficiently the indications in meningococcemia, and thus are very useful where the patient is vomiting and not able to take the drug by mouth. Some sulfonamides are more absorbable in the spinal fluid than others. Sulfanilamide is the most absorbable; sulfapyridine is next, with rapid concentration in the spinal fluid of 65 per cent of that of the blood. Sulfadiazine also gives a 65 per cent concentration in the spinal fluid, but is slower in building up the titre. Sulfathiazole is least absorbable, diffusion is delayed and final concentration is low. Concentrations of sulfamerazine are comparable to those of sulfadiazine in the spinal fluid, but perhaps a little below. The figures given are 50 per cent of that in the blood serum.

A series of cases, reported by W. I. Gefter *et al*,[6] in the *American Journal of the Medical Sciences*, treated with sulfamerazine gave a mortality of 6.7 per cent. Sulfamerazine concentrations of 10 to 15 milligrams per cent per 100 cc. is considered adequate. The sulfa drug should be continued for 72 hours after the return of temperature, pulse, and respiration to normal. This blood titre can be accomplished in children three to ten years of age by 1½ grams initial dose and 1 gram every 12 hours; in infants six months to three years by 1 gram initial dose and ½ gram every twelve hours; and in infants under six months by ½ gram initial dose, and ¼ gram every 12 hours. In severe infections this dose may be increased 50 per cent. In the case of pneumococcic and streptococcic infections as well as meningococcic infections, if the patient does not show definite improvement within 24 hours, the specific antisera should be administered also. Such conditions are more likely to occur in the infant, in the aged, and in the otherwise sickly child.

In my experience, the best results have been obtained by the combined sulfa and serum treatment. Sulfamerazine has been used successfully in epidemic meningitis in pregnancy, without harm to the fetus.[7]

My experience with primary pneumococcic meningitis, which I have seen mostly in the infant, has been very unfortunate. In spite of the use of specific antipneumococcic serum and sulfapyridine and sulfadiazine, all of my cases have died. But there are case reports in M. D. literature of all surviving. However, with influenzal meningitis, conditions have been improved by the use of the combined sulfapyridine or sulfadiazine and the specific rabbit influenza serum to where the mortality has been reduced from 100 to about 50 per cent. A series of 50 cases treated by sulfanilamide and rabbit serum were reported by Alexander, Ellis, and Leidy,[9] with a mortality rate of 26 per cent.

Influenza bacillus meningitis is not uncommon in children. The treatment of influenza meningitis in a series of 15 cases is reported by Lamm and Shulman.[9] As a result of their experience, they have outlined a technic of treatment which they think gives the best results. It consists of the intravenous and oral administration of sulfapyridine, and the intravenous administration of anti-influenzal rabbit serum. Blood antibody titres are measured at intervals by means of a capsular swelling test on *H. influenzae* with the patient serum. "A positive capsular swelling reaction with a dilution of one to ten or more indicates adequate amounts of circulating antibodies." Sulfadiazine and sulfathiazole are used in place of sulfapyridine, where the response is poor to the last named. Blood transfusions are used as supportive measures. They state, as we all have learned who have treated cases of influenzal meningitis, that there is a great tendency to exacerbations and relapses so that chemotherapy should be continued for two to three weeks after the spinal fluid is sterile. Bloomberg, Tannenbaum, and Glich[10] report eleven cases of influenzal meningitis treated with sulfa drugs and anti-influenzal serum. Those cases that they saved received sulfapyridine and sulfadiazine intravenously and by mouth. Where sulfanilamide alone was used, with or without influenzal serum, the patients died. Their mortality rate was 45.4 per cent. Alexander and Leidy[11] conclude that sulfadiazine is superior to sulfanilamide and sulfapyridine in mouseprotected capacity. They conclude that "in the treatment of patients with influenzal meningitis the risk of failure is reduced to a minimum by the use of both serum and sulfadiazine."

CONVALESCENT HUMAN SERUM THERAPY

I think is is proper that some of the diseases to be discussed in this article should be handled under the heading of convalescent human serum theapy. Dr. C. M. Hyland of Los Angeles has been a pioneer in developing the place of convalescent human serum in many acute contagious diseases. Convalescent serum is now being accumulated, titred, packaged, and shipped from several serum centers in the United States. The one in Los Angeles, conducted by Dr. Hyland, is the one with which I am familiar. Here are prepared convalescent measles, scarlet fever, mumps, chicken-pox, poliomyelitis, and pertussis sera. Their value is variable, ranging from very effective in measles and scarlet fever through the moderately effective ones such as mumps and chicken-pox to the questionably valuable poliomyelitis serum. Convalescent pertussis serum probably ranges with mumps and chicken-pox sera in value. My personal experience has been rather considerable in private practice with their use. Human sera have one particular advantage over sera taken from horses or rabbits, in that there is little or no reaction whatever and no danger from anaphylaxis or serum sickness.

Measles—Long before convalescent serum was used in measles, I used to use whole blood infusions intramuscularly from measles convalescents in the prophylaxis and treatment of measles. Early in an epidemic period, I would find some healthy older child or young adult, who had recently convalesced from measles, and who had a good blood count and was free of tuberculosis or syphilis. I would take him with me to the home of the patient who was exposed or sick with measles, and give the patient some 20 to 30 cc. of whole blood intramuscularly. This was equivalent to 10 or 15 cc. of serum. I had quite satisfactory results in both preventing and modifying the course of measles.

Measles convalescent serum is used both in prophylaxis and modification of measles. A preventive dose should be given as close to exposure as possible, certainly not later than five days, and before the appearance of symptoms. After symptoms have appeared, of course, it is possible only to modify the disease. When given four or five days following the date of exposure, the recipients usually do not develop measles. Since the percentage of deaths from measles in infants under two years of age is quite high, even the temporary passive immunity lasting for three or four weeks is sufficient to protect them and is highly desirable. A child under two years of age can be protected at the time of exposure with 5 cc. of convalescent serum. If exposure occurs again in two or three weeks, he should be injected again. When the patient is older and in good health, it is better to modify the disease than to prevent it. This is accomplished by injecting 7½ cc. on the sixth or seventh day after exposure, to children from three to ten years of age. In the pre-eruptive stage, but after the appearance of Koplik's spots, the intravenous injection of 50 cc. of serum is necessary to modify the disease. This would be of value and desirable in the case of a sickly tuberculous child or one in whom the disease is manifesting unusual toxicity or severity. This should be done also in children who have just recovered from another acute infection, such as pertussis.

However, reports are current of the modification of measles even in the eruptive stage with intravenous doses of from 10 to 20 cc. The serum given during the eruptive stage modifies the intense incessant cough, and the temperature drops. The rash and symptoms clear up quickly. Most pneumonias and middle ear and mastoid infections in measles are caused by hemolytic streptococci. When these complications occur in measles, it has been suggested that scarlet fever convalescent serum be administered intravenously.

Scarlet Fever.—We are all familiar with the clinical manifestations of scarlet fever and its complications, otitis media, mastoiditis, meningitis, hemorrhagic nephritis, endocarditis, et cetera. It is known to be caused by the beta hemolytic streptococcus. There are a number of different strains of this organism, all of which can produce a scarlet fever rash at times, but may not always do so; when they do not it is called scarlet fever sine eruptus. Cross immunity between these strains is always present, but not always complete. Many of the symptoms of the disease are produced by a soluble toxin, yet the organism itself may

and often does invade the blood stream of the patient. Other diseases caused by the beta streptococcus and giving a certain amount of cross immunity to scarlet fever are streptococcic sore throat, erysipelas, and puerperal sepsis.

The sulfonamides used early in the disease or for complications are effective in that they have a definite bacteriostatic action, but they do not have any antitoxic effect. Therefore, the convalescent serum can supplement the sulfonamides very nicely and adequately in the severe and toxic cases of these infections. The scarlet fever convalescent serum can also be used in prophylaxis among contacts in a family or in an institution. It does not have the dangers inherent in the sulfonamides. It can be used during the course of the disease as well as prophylactically, and it is very effective in controlling all the toxic manifestations such as fever and malaise. The local symptoms in the nose and throat are also benefited, and complications are reduced to 50 per cent of their usual rate. As said before, there is no anaphylaxis and no sensitization. The dose is from 20 to 100 cc. intravenously, depending upon the course of the disease and the degree of toxicity. In a paper presented at the Rocky Mountain Medical Conference, Dr. Hyland[12] reports good results in infectious mononucleosis with the use of 100 cc. of scarlet fever convalescent serum given intravenously.

Another use of the convalescent scarlet fever serum is that of diagnosis in producing the Schultz-Charlton blanching phenomenon. Like all other sera it should be administered intravenously and early in the disease. In our work at the County Hospital, sulfa drugs rather than convalescent serum are used in the treatment of erysipelas because of the expense of the serum. But in private practice, in an infant under one year of age that is so likely to develop the phlegmonous form, I would certainly use them both. Certainly the convalescent serum should be used in cases where a sulfonamide is contraindicated. Less of the sulfonamides can be used when the convalescent serum is given. Later penicillin may be used in the treatment of pneumostreptococcic infections more commonly than the sulfonamides and may replace the latter, but it cannot replace a specific antitoxin.

Mumps.—Less valuable than measles or scarlet fever convalescent serum is mumps convalescent serum. I have had very good results, but also some failures in the prophylactic use of mumps serum. The dose recommended is 20 cc. intravenously (40 cc. for large children) soon after exposure. Recently I have had two cases of complete failure of the serum to prevent mumps. However, in a series of cases reported by Thalhimer[13] of the Manhattan Convalescent Serum Laboratory, in a total of 84 cases using the recommended dose, 83 were prevented. Five others with insufficient dose were modified. I have had spectacular results in mumps meningitis and mumps orchitis in the relief of pain and the subsidence of the temperature within twenty-four hours. I have also had equally good results with convalescent whole blood.

About five years ago a young girl ten years of age recovered from an uncomplicated case of mumps. Her mother developed mumps meningitis as indicated by characteristic spinal fluid findings plus the presence of parotid swelling. After three intramuscular injections of 20 cc. of blood, taken from the child and given to the mother during a period of twenty-four hours, the symptoms of meningitis quickly subsided, the headache and delirium left, and the temperature dropped to normal.

In spite of some failures, therefore, I am very enthusiastic about its use.

Chicken-Pox.—Dr. C. M. Hyland[12] states that convalescent serum of chicken-pox has a 40 to 50 per cent protection rate in exposed patients, provided it is administered in sufficiently large doses. Encephalitis occurs occasionally in chicken-pox. Dr. Hyland cites a case which improved in a few hours after the intravenous administration of 650 cc. chicken-pox convalescent serum. The paralysis or hemiplegia that follows chicken-pox is likely to recede spontaneously, but only over a considerable period. There might be some tendency to epilepsy, as there is in the encephalitis following other communicable diseases. If one could be reasonably sure of treating such a condition successfully, it would be well worth the expense.

Pertussis.—Whooping cough today is successfully prevented in a very high percentage of instances by the use of vaccines and other substances. Of these there are the phase one concentrate and super-concentrate made by Lilly and Cutter, the pertussis antigen made by Lederle, and the combined diphtheria and pertussis alum precipitated toxoid. The successful use of these substances in treatment is in question. Personally I have convinced myself of their value. I prefer the pertussis antigen. I feel that its use shortens and makes more mild the course of the disease. Recent studies indicate that in efficacy of prevention determined by the complement-fixation tests, pertussis antigen, twenty billion per cc., a total of 5 cc. or one hundred billion organisms given in three doses at monthly intervals, gives the best results; with alum precipitated toxoid, a total of forty billion in one injection, next in efficacy.[14]

Infections by *Bacillus bronchisepticus* and *Bacillus parapertussis* give pertussis-like coughs. They can be differentiated only by cultural methods; nevertheless the cough plate is the first step in the diagnosis. A total high white count with lymphocytes predominating usually develops during the paroxysmal stage which is too late for either prophylaxis or treatment. Antibodies do not develop in the blood until too late to be of practical value in treatment. Clinically there are cases of atypical pertussis without the whoop, particularly in infants and adults or children incompletely immunized. Catarrhal tracheobronchitis may simulate whooping cough clinically. Therefore one cannot depend today upon clinical differentiation. The cough plate is therefore the best means of early diagnosis. Naturally a reasonable certainty of diagnosis is desired before convalescent serum is given. Convalescent serum has the most value in little infants and sickly children, particularly those with tuberculosis or with some cardiac weakness.

Hyperimmune rabbit serum is now available. I have had some satisfactory response from its use.

Pertussis hyperimmune human serum is prepared by immunizing healthy human beings with vaccine. The results with this serum have been very satisfactory. A report on 254 cases at the Philadelphia Serum Exchange gave 96 excellent results, 69 good results, 55 moderate results, 29 poor results, and 5 deaths. Infants less than six months of age were injected with 20 cc. of serum every other day for three doses, the

more severely ill receiving additional doses. The best results occurred when serum was given before the third week of the disease.[15]

Poliomyelitis—Generally convalescent serum in poliomyelitis has been discredited. Personally my experience with it has been rather satisfactory. I have had a number of cases, including my son, where the results have been spectacular. I would use, however, no less than 50 to 100 cc. intravenously in one dose, and of course the earlier it is given the better the results. Some pools of serum show an almost complete absence of neutralizing antibodies, but other pools have very high neutralizing value. If the antibody titre was always determined, and only serum of high titre used, I feel that much better results could be secured. Dr. Hyland states, "I am still enthusiastic about poliomyelitis serum."[16]

STAPHYLOCOCCIC SEPSIS

I wish to discuss some of my experiences in the handling of staphylococcic sepsis at the Los Angeles County Osteopathic Hospital. Ten years ago, a male patient, seven years of age, came into the Children's Ward with the following history: He fell off a stone wall August 20, 1934. On August 28, he developed sore throat and nausea, and pain in his left thigh. His temperature rapidly rose to 104.4 F., and he developed tenderness and swelling of the left thigh muscles with external rotation and with an edema and erythema of the medial aspect of the left thigh. His tonsils were enlarged and inflamed. A blood count showed erythrocytes 3,950,000, leukocytes 18,200, polymorphonuclears 86 per cent. He was admitted to the hospital August 29. A blood culture was taken and on August 31, a positive report of Staphylococcus aureus was made. X-rays of the femur September 1 revealed normal bone, but x-rays taken September 3 showed rarefaction of the femur below the lesser trochanter. Diagnosis of osteomyelitis was made and an operation decided upon. He died shortly following surgery. The autopsy showed osteomyelitis of the right femur, bronchial pneumonia, acute tonsillitis, and hemorrhagic spleen.

This case was seen before the era of adrenal cortex extract, vitamin C, and blood transfusions, and subsequently staphylococcic antitoxin. When using staphylococcic antitoxin with repeated blood transfusions, adrenal cortex hormone, and large doses of vitamin C, we have saved the lives of many such cases. Some have had staphylococcic pneumonia and empyema and multiple areas of osteomyelitis, and have still lived. Since the use of sulfathiazole in large doses administered both intravenously and by mouth in pushing the blood titre up to 20 to 30 milligrams per 100 cc., I have seen even better results, In such cases recovery has occurred even with extensive pulmonary involvement to the extent that the patient had to be kept under oxygen for two weeks, but recovered without surgery and without any sequellae, the affected limbs being immobilized in casts without drainage. Staphylococcic antitoxin has been used in conjunction with sulfathiazole in several instances. No patient has died where we have attended him early, and two patients survived that we thought were moribund.

We have had little opportunity to try penicillin in these cases of staphylococcic sepsis. The last patient we had was an unusually well-developed girl eleven years of age. She had recovered from scarlet fever one week previous and during the last five days had had several staphylococcic abscesses on her right leg. She had been given sulfadiazine every four hours, grams ½, for the last four weeks by her doctor. The dose was very inadequate. On admittance, she was cyanotic and dyspneic, with a fever that was septic in character. The right leg was swollen and edematous to the thigh, resembling milk-leg. Her pharynx was red and inflamed. There were sticky moist fine rales on inspiration over her chest. Pelvic examination revealed normal organs. The blood count was 3,500,000 red and 28,000 white blood cells and 80 per cent polymorphonuclears. The blood culture was positive for Staphylococcus aureus. A blood titre was maintained for one week of 10 milligrams per cent of sulfadiazine, (not enough in my opinion, as her blood culture remained positive). Her urine showed two plus albumin and some granular casts and no red blood cells. During the course of her illness she was given sulfathiazole and sulfadiazine both intravenously and by mouth. During the administration of the sulfathiazole, staphylococcic antitoxin was given to the extent of about fifty thousand units intravenously. (Neither sulfathiazole nor antitoxin was given in adequate doses.) The sulfathiazole was then discontinued, and penicillin started, subsequent to which she improved clinically, and the blood cultures became sterile. An abscess of her right tibia was opened and drained; and abscesses over the sternum also was drained. Bilateral thoracentesis was performed and several hundred cc. of staphylococcic pus removed. A total of 1,396,000 units of penicillin were given. Repeated blood transfusions were given, of from 150 to 250 cc. each. A high vitamin C intake was maintained. After no more penicillin was available to us, the sulfathiazole was resumed, reaching a blood titre of 5½ milligrams per 100 cc. She had received continuously throughout her stay in the hospital oxygen by intranasal catheter. She died, and the autopsy did not disclose osteomyelitis.

This case was in contrast with some others previously handled by sulfathiazole and antitoxin alone that seemed at first to have as extensive lung involvement, but which recovered. I feel that if this patient had had even larger doses of sulfathiazole and antitoxin, or still larger amounts of penicillin, she might have been saved.

Of course, the literature today is full of staphylococcic bacteremia and sepsis being cured by the sulfonamides; and where in certain instances the staphylococcus is sulfonamide-resistant, the patient is being cured with penicillin. Such a case was reported in the *Journal of the American Medical Association*, December 11, 1943, wherein staphylococcic antitoxin was not used and wherein the highest sulfathiazole blood titre was seven milligrams per 100 cc., and the patient was cured by the use of a total of 540,000 units of penicillin sodium. The experience, then, is overwhelmingly in favor of the value of penicillin in the sulfonamide-resistant infections of the staphylococci, also in gas gangrene, hemolytic streptococcic infections, pneumococcic, gonococcic, and meningococcic infections not responding to the sulfonamides.

TETANUS

Tetanus is one of the most gruesome of diseases. We have several cases each year on the Pediatrics Service of the Los Angeles County Osteopathic Hos-

pital. These cases of tetanus are mostly in older children, particularly boys. We have never had a case, so far as I can remember, of tetanus neonatorum, either on the general wards or the surgical wards. This is prevented by obstetrical asepsis.

Prophylaxis is the ideal treatment. Up until recently, prophylaxis consisted of administering from 1,500 to 3,000 units of tetanus antitoxin intramuscularly following any wound wherein there existed the possibility of contamination with the tetanus spores or bacilli. This procedure has no doubt saved many a case of tetanus from developing and many a life consequently.

Some individuals are extremely allergic to tetanus antitoxin. I have seen cases of angioneurotic edema, arthritis, and laryngeal edema from its use, and a high per cent of the cases develop urticaria which persists for several days and is very annoying. The administration of tetanus antitoxin should always be preceded by tests for hypersensitiveness which, if present, calls for desensitization, which can at times be tedious and time consuming. A very high per cent of children suffering from penetrating wounds would never develop tetanus anyway. We occasionally run across a child with tetanus where there is no history of any wound or abrasion that would suggest the need of antitoxin.

It has long been recognized that a prophylactic procedure relatively without danger, and which could be given routinely to all children, much as immunizations against diptheria are given, would be highly desirable. This has been found in alum precipitated tetanus toxoid. This preparation is put out by several biological houses. Two injections are usually given of from ½ to 1 cc. and repeated in two or three months. Alum precipitated tetanus toxoid may be combined with diphtheria-pertussis alum precipitated toxoid, in which instance three doses are given at monthly intervals, of ½ cc., 1 cc., and 1 cc., respectively.

As a consequence of these injections a variable degree of tetanus antitoxin will circulate in the blood of the patient. Gradually over a period of time, perhaps six months to a year, the blood titre drops below the protective value; but if at any time it is needed, another injection of alum precipitated toxoid may be given and will very rapidly increase the blood titre to a protective degree, just as though the antitoxin itself were given.

If, however, the patient actually acquires tetanus, then comes the problem of saving the child's life. Let us remember the early symptoms of tetanus. They are restlessness, insomnia, irritability, headache, meningeal signs, soreness, stiffness and tonic painful spasms of the pharynx, and of the back, neck, abdominal wall, and face, such as trismus or lockjaw, and the fixed painful smile called risus sardonicus. There are also spastic gait, hyperactive reflexes, anuria, and convulsions. Rigid abdominal muscles are sometimes one of the first symptoms. The first symptom noted in infants may be convulsions. The incubation period is from one to thirty-five days; the shorter the incubation period the higher the mortality rate. Sulfanilamide dusting powder has been known to contain tetanus spores.

Once tetanus has been diagnosed, active and vigorous treatment must be instituted immediately. I am quoting verbatim the orders as outlined in the Los Angeles County Hospital House Staff Manual:

ACTIVE TREATMENT

1. *Sedation:* started as soon as diagnosis made. Sedation must be sufficient to allow patient to rest or sleep, but should not keep him in state of complete narcosis or anesthesia because of danger of pneumonia.

(a) Chloral hydrate and calcium bromide, 15 to 30 grains each by mouth, or in an ounce of water by rectum. Repeat as necessary, adjusting dose to patient.

(b) Barbiturates by mouth or subcut. Sodium luminal gr. 2 to 4 intravenously for severe convulsions.

(c) Avertin rectally.

(d) Keep in darkened, quiet room. Avoid sudden noise or bright lights.

2. *Local treatment* is deferred only 30 to 45 minutes to give opportunity for sedation to take effect.

(a) Extensive débridement of focus, wherever located, is at present considered an emergency measure to be performed as soon as possible.

(b) Cold 1:600 potassium permanganate compresses may be applied constantly to the wound after débridement if conveniently located.

3. *Antitoxin*

(a) Skin test and intravenous test for sensitivity.

(b) 40,000 units injected around the wound prior to débridement.

(c) 40,000 units intramuscularly injected in an encircling manner about proximal portion of involved extremity if wound is on extremity. If site is unknown or internal, 60,000 to 80,000 units may be given intramuscularly.

(d) 40,000 intravenously in 300 to 500 cc. normal saline, slowly, preceded by 5 minims of adrenal extract. Intramuscularly and intravenously to precede débridement.

(e) Repeat 40,000 units of antitoxin intramuscularly in 8 to 12 hours after first dose.

(f) Total dose usually 160,000 to 200,000 units. Variation in dosage and routine at the discretion of the staff.

(g) 1,500 units every fourth day as long as patient remains in the hospital (for desensitization).

4. *Diet* as tolerated. Liquids usually best. Use rubber drinking tube. Never put glass tube between teeth. Fluids parenterally if necessary.

5. *Cause of death* sometimes uremia. If in doubt, check blood-nonprotein nitrogen.

6. *Artificial respiration,* prone pressure method, if respiration stops in a convulsion.

7. *Check x-rays* of thoracic and lumbar spine before discharge.

Comment.—Let me emphasize the statement that sedation must be sufficient to allow the patient to rest and sleep, but be short of narcosis. The patient has to be watched closely enough so that sedation may be given as often and in as large a quantity as necessary. It is surprising how large a quantity of strong sedatives a child will require in the active stage of tetanus. Patients are known to recover with sedation alone, through production of their own antibodies. On the other hand, no amount of antitoxin will save some patients where a considerable amount of the toxin has become fixed in the nervous system in spite of sedation. If sedation is not used, the effects of fatigue and its toxins are added to the toxemia of the nervous system. As is well known, it is very important to prevent convulsions in order to avoid fractures of the spine that occasionally occur. Obviously the earlier antitoxin in large doses is administered the better the prognosis, as it is essential to neutralize the toxin quickly and completely in as short a time as possible to prevent its fixation in the nervous system. The dosage of antitoxin as given in the Manual is sometimes not enough in severe cases. Let me emphasize also by repetition that it is just as essential as sedation.

in fact it is part of the sedation technic, to keep the patient in a darkened, quiet room and to avoid sudden noises or bright lights. With this technic we feel that our results have been very satisfactory.

X-RAY THERAPY

Time will not permit a detailed or extensive discussion of the use of x-ray in the treatment of the infectious diseases in children. In passing, we are reminded that it is being used in the following conditions: Acute virus or atypical pneumonia, other acute bacterial pneumonias, and bronchiectasis. Then, too, whooping cough is often modified beneficially with one, two, or three x-ray treatments. I have never seen a case aborted, and I have seen cases that apparently were not helped at all. I think the type of case which is helped the most is that wherein there is a considerable lymphatic hypertrophy in the parabronchial and mediastinal lymph nodes. Asthma is being helped in some instances by x-ray treatment. I cannot as yet feel sure of its value, as psychic factors enter into the asthmatic case to such a great degree. Acute and chronic osteomyelitis are many times treated very effectively by x-ray, and this should be given a thorough trial. The x-ray treatment of enlarged thymus is an old, established procedure. In cases where surgery is contraindicated as dangerous, x-ray may be used to reduce adenoid vegetations in the nasopharynx in the infant and small child, and thus relieve obstruction.

ATYPICAL PNEUMONIA

Virus or atypical pneumonia is very prevalent in our part of the country as it no doubt is elsewhere. In the days before the sulfonamides, its virus nature was perhaps not recognized, and it was diagnosed as bronchial pneumonia with a tendancy to relapse, or it was diagnosed as influenza bacterial pneumonia, but thought to be due to secondary bacterial invaders. Sulfathiazole, sulfadiazine, and sulfamerazine have been used in many cases of atypical pneumonia, which have been found not to respond to them as the bacterial types of pneumonia do. The causative virus has been recognized and demonstrated by studies on special cultural media, by neutralization technic, and by the demonstration of inclusion bodies in the bronchial epithelium. The clinical blood count and x-ray findings are fairly well established. Clinically, a rather gradual onset with at first a normal or leukopenic blood response and a nonproductive cough that may be rather spasmodic are uniform characteristics. The fever is quite variable, and may fluctuate from two to three degrees in twenty-four hours. Varying degrees of cyanosis may be present; and in severe cases, the cyanosis and dyspnea is extreme, as in capillary bronchitis and suffocative catarrh. The x-ray picture is characterized by patchy diffuse shadows in the lung fields that are inclined to change location, and further may be characterized by a heavy hilar shadow extending into the interlobar fissures with soft mottling into the bases of the lung.

When, later in the disease, eosinophilia occurs, as it often does, it makes one think of Loeffler's syndrome, which is recognized as an allergic phenomenon. I have often found eosinophiles present in the blood count throughout the course of the disease, and to me it is one important diagnostic aid in differentiating from bacterial pneumonia. The x-ray findings may be increased all out of proportion to the physical findings on auscultation and percussion. Areas of bronchial breathing are occasionally found, as are fine crackles at the end of inspiration; but very commonly the findings characteristic of consolidation are modified by areas of emphysema, so that the percussion note is tympanitic rather than dull, and the shadow may not be seen on the x-ray plate.

It is not possible at present to use virus studies as a practical clinical means of diagnosis; therefore the diagnosis is based upon the clinical, x-ray, and laboratory findings, particularly the blood count. A therapeutic test for forty-eight hours with the sulfonamides may be justifiable. I emphasize again that the blood count is very helpful for diagnosis, as acute bacterial infections give, in the early stages, a much higher white count, that is from 18,000 to 20,000, as compared with the virus infection of from 6,000 to 10,000. Also, eosinophiles are likely to be present in the blood count in the virus type, while they are uniformly absent in the bacterial type. I have found myocarditis, hepatitis with enlarged liver and subclinical jaundice, and enlarged spleen frequently in severe cases of virus pneumonia. The liver and spleen involvement is reflected in the blood count in an increased number of mononuclears.

There is as yet no specific treatment for virus or atypical pneumonia. However much can be done to relieve the symptoms and shorten the course. The milder cases recover readily with complete and prolonged rest in bed and osteopathic manipulative treatment. In children blood infusions from older members of the family who may have recovered recently from the same virus infection without having had a pneumonia are very helpful. Possibly convalescent serum, if it were available, would be definitely curative. For the prolonged and persistent spasmodic cough, I have used medicated steam inhalations, the iodides as potassium iodide and calcidine, short-wave diathermy, and intramuscular injections of ether and camphor in oil. The last named I have found quite efficacious in relieving bronchial spasm and liquefying the thick mucus. I also use these injections in whooping cough and whooping cough pneumonia. I have never relied entirely on osteopathic manipulative treatment in these cases, but I find it to have a very definitely beneficial effect.

Oxygen must be used in the more severe cases with cyanosis and dyspnea. I have seen patients wherein high concentrations of oxygen were insufficient to relieve these symptoms. Intravenous glucose is often very helpful in protecting a toxic liver and supporting a weak myocardium. It should be given in a hypertonic solution. Finally x-ray treatment is reported as being very efficacious. As yet I have had little experience with x-ray as a treatment, most of my patients having been cared for in the home where x-ray was not available. When, later, the hospital situation with the great shortage of hospital beds is corrected in our vicinity, I intend to take my more severe cases, when possible, to the hospital and try out the x-ray treatment. We have had several patients on the Wards of the County Hospital that have responded very satisfactorily to x-ray treatment.

I intended in this paper to discuss coccidioidal disease of the lungs, which is being written up in recent literature rather extensively and which is very prevalent in California, so prevalent in fact that it

is called California disease. It is also called coccidioidosis and coccidioidomycosis. It is endemic in the Southwest and is caused by the *Coccidioides immitis*, an organism belonging to the yeast group and closely related to the blastomycetes. The subject deserves more space than I have to spare. For those that are interested, let me recommend the section on coccidioidal granuloma in "Diseases of the Chest" by Norris and Landis, sixth edition, and the article on coccidioidomycosis by Charles Edward Smith, M.D., in *Medical Clinics of North America*, May, 1943. The latter article is very complete with a large bibliography but without extensive statistics or case histories.

Poliomyelitis is another subject I have not room to discuss. There is so much new material in the literature, based upon clinical and pathological studies, stimulated by Sister Kenny's work, that to do justice to the subject a separate paper would be required.

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The Problem of Infection in the Chronically Ill Child*

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Our opinion may be expressed in one sentence: Infection in the chronically ill child, and for that matter in adults too, is never primary. It is at least secondary or tertiary, frequently even more remote. The present laboratory concept of infection and immunity necessitates relegation of infection in the causation of disease to a subordinate role. Clinical observation has confirmed this. We believe also that the word, "disease," as commonly used is incorrect. Solis-Cohen[1] says that "Disease is the struggle for health." We believe that this dynamic process cannot be circumscribed by the usual restrictive terminology.

A search for the definition of the word, "infection," reveals no established interpretation. Definition varies with the dictionary or text and its date of publication. Several basic factors seem to appear in these references with sufficient constancy to allow for the establishment of five elements. These are:

1. Invasion of the body tissues (by)
2. Pathogenic microorganism (which under)
3. Favorable circumstances (such as)
   (a) Overwhelming numbers (or)
   (b) Virulence (or)
   (c) Lowered resistance of the body—or all three—(permit)
4. Multiplication of the microorganisms (with)
5. Local or general reaction or both.

Evaluation of these five elements enables us to interpret more accurately the status of infection.

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Invasion of the Body Tissues.—Walter B. Cannon,[2] Professor of Physiology at Harvard Medical School, reminds us that, "The most potent immediate protection against these harmful forms [of bacteria] is an intact and healthy body surface. Only when the skin is broken or cut, are the conditions favorable for the invasion of our bodies by these foreign enemies." Barring an actual break in the skin or mucous membrane surface, we are not attacked by microorganisms except in the presence of a weakening of the integrity of the body's surface. This may be considered a departure from the "healthy body surface" protecting us from microorganism invasion. Examples given by Cannon[3] are: " . . . failure to keep a part clean, or . . . slight injury or some general weakened state, pus-producing bacteria, which are living close to the root of a hair in the skin, begin to multiply rapidly."

Pathogenic Microorganisms.—We are concerned here only with pathogenic microorganisms. As a sterile world is at present infeasible and bound for prompt extinction, according to bacteriologists, this requirement need not be discussed.

Favorable Circumstances.—

Overwhelming Numbers of Bacteria: Sanitation, isolation, quarantine, asepsis and antisepsis are among measures taken to remove or reduce this element. As large numbers of almost pure cultures of microorganisms are frequently available in an infected source, we attempt to prevent inoculation from such a place. This is part of preventive medicine.

Virulence: According to Jordan and Burrows[4] "virulence" is difficult to define. They attempt it, how-

ever, by stating that it is "the ability of a bacterium to produce disease." The known properties contributing to virulence are:

(1) Toxins; (2) the "invasiveness"; (3) hemolysins; (4) leucocidins; (5) coagulase and fibrinolysin; (6) "spreading factor" of Duran-Reynals; (7) capsules; and (8) miscellaneous factors such as pyogenesis, necrotoxin, hypothermic factor, edema-producing factor. These are of importance to us here only in that we know that each of them acts as an antigen with the production of specific antibodies to neutralize or counteract its activity—this, in the healthy body.

Lowered Resistance of the Body: As the resistance of the body is dependent upon several factors, it seems best to list the following more important ones: (1) Inherited resistance; (2) species and racial resistance; (3) climate and seasonal factors; (4) external body defense (i.e. skin and mucous membrane); and (5) general well-being of the body. Jordan and Burrows[6] consider the factor of general well-being of "profound importance" in the body's resistance to infection. They consider general well-being dependent upon: (1) Presence or absence of previous infections, which may prepare the way for secondary infection by a lowering of the body's resistance; (2) presence or absence of functional disorders, such as diabetes; (3) nutrition; and (4) fatigue. To this brief list as given in their textbook may be added the many factors, so often slighted, which may be considered the effect of the environment on the psychic make-up of the individual.

Boyd[6] states that resistance depends upon three great factors: (1) Humoral antibodies (antitoxins, bacteriolysins, opsonins); (2) phagocytosis; and (3) the immunological (i.e. chemical) behavior of certain tissue cells. As all of these last named factors are constituents of the healthy "fluid matrix" of Cannon, we must remember A. T. Still's,[7] "The rule of artery and vein is universal in all living beings . . . "

Multiplication of Microorganisms: This begins immediately upon entrance of a microorganism into an environment of favorable characteristics, as most bacteria reproduce themselves every twenty minutes. However, if the environment is not favorable or is made unfavorable by either internal (homeostatic) mechanisms, or by external (antiseptic) procedures, multiplication is quickly halted. Internal or homeostatic mechanisms are dependent upon an adequate supply of healthy blood, or as Cannon would put it, maintenance of the "fluid matrix."

Local or General Reaction or Both.—Boyd[8] lists the results of infection as follows: (1) Local inflammatory lesion at the site of entry which is always present although it may be microscopic; (2) spread by means of septicemia, bacteremia or pyemia; and (3) toxemia.

The toxemic changes in the body tissues are "degenerative in character, ranging from cloudy swelling and fatty degeneration to necrosis and disintegration of the cells."[9] Toxemias of long duration may result in "amyloid degeneration." "Fever, loss of strength, wasting, increased BMR, and leucocytosis are evidence of the effect of toxins on the body."[10]

Forbus[11] states that "disease" of any kind is to be viewed as the essential reactions of the individual (in the last analysis of the individual cells) to agents and influences comprising the environment. The cell may (1) resist, (2) submit or (3) effect an adaptation. The generally recognized "disease" entities are considered to be expressions of these three reactions. When we review the elements of infection there appear to be two items of interest to the chronically ill child problem: (1) The symptoms of toxemia which are found so frequently, and (2) a lowered resistance which seems to be the outstanding factor necessary for subjection of the body to the ravages of infection and of illness.

We feel that a lowered resistance is first necessary—the causes of which are limitless; then, secondarily, that microorganism invasion with resultant symptom-complexes develop. In the presence of a lowered resistance, acute, subacute and chronic illness may be evolved.

From the standpoint of infection as considered in this paper, we must include the latent or hidden infection which in mechanism is the same as the cryptic infection. In both of these, virulent microorganisms have invaded body tissues and are held in check by the body's defenses. When resistance drops further a "flare-up" results. Here is the focus of infection, so frequently a point of argumentation. If we remember Boyd's summary of toxemia we shall be diligent in our search for foci of infection and insist upon their removal.

Foci of infection are requently the rearguard of a previous infectious illness. This rearguard serves as the point of a counterattack launched when the body's defense weakens. Through their presence they effect a constant drain of the body's energies not only to keep the microorganisms in abeyance but also to combat the toxins and repair tissues damaged by both organisms and toxins. Proper convalescent and postconvalescent care is the preventive means.

Foci of infection in the child are sometimes difficult to locate. Whether actual infection is present in some conditions, or congestion only, is controversial. Tonsils, about which much discussion has been had, are in our opinion the most frequent source of toxin production in the child. Pus, to us, denotes infection; infection affects the general body, not only by definition but also as evidenced by bacteriological studies of the opsonic index, agglutination tests, antitoxin formation and serial Schilling differential blood studies.

Tonsil infection is usually the result of a streptococci or staphylococci, or mixed invasion. Both of these microorganisms produce exotoxins. The acute tonsil infection with septicemia and pyemia may produce endocarditis. The chronic or cryptic tonsil infection so often allowed to remain for many years, will, as Boyd has told us, produce over a period amyloid degeneration by means of a chronic toxemia. Degeneration, a "change from a higher to a lower form; especially change of tissue to a lower or less functionally active form"[12] produces many symptom-complexes. Depending upon the organ or organs presenting predominant symptoms, we plan our treatment. This is incorrect. We are ignoring both the secondary or tertiary factors of infection, and the other organs involved to a subclinical degree. Is it any wonder that the chronically ill child is such a problem, if infection alone presents such a silent stumbling block to our diagnostic and therapeutic efforts?

In our practice we strongly recommend tonsillectomy in the presence of any one of the following findings: (1) Enlarged tonsils, (2) inflamed tonsils, (3)

history of tonsillitis, (4) extrusion of pus on pressure, and (5) cervical lymphatic gland enlargement and toxic cervical musculature, in the absence of other head infection.

Congestive states of the adenoids or nasal mucous membrane lower the resistance of those membranes to microorganism invasion and allow establishment of foci of infection. How frequently actual microorganism invasion occurs we know not; however, we do know from the literature on bacteriology that microorganism invasion is said to occur in the nasal passages. The frequent illnesses of the child may result from the congestive state found to be present. It is our opinion that adenoidectomy is indicated.

Nasal accessory sinuses have been discussed by Looper[13] of the University of Maryland, who says, "It is a common practice to attribute all chronic colds and all cases with nasal discharge, regardless of location, character or amount of secretion, to diseases of the tonsils and adenoids. It is often taken for granted that this is the only factor to be considered, while in fact the true source of the infection may be in the paranasal sinuses." One reason why we have tended to overlook this factor is the misconception that the sinuses in children are so undeveloped that they are not a causative factor of early nasal infections. Onodi, Davis and Schaeffer have studied fetal, newborn and child skulls from six and a half months prenatal to nineteen years of age. The frontal sinus makes its appearance from the end of the first year to the beginning of the third year. The sphenoid sinus is a faint depression at birth. Its real development begins at the end of the fourth month. The ethmoid cells are present in the newborn. The maxillary sinus shows rudiments before the middle of fetal life and begins to grow about the second year.

Folds or reduplication of mucous membrane in these developing sinuses may be likened to tonsillar crypts in that they are ideal locations for growth of bacteria—on the membrane surface. As discussed previously, lowered resistance or a break in the membrane may result in microorganism invasion. This may be the explanation of the widespread sinusitis, so resistive to treatment, in our adult population. Wachsberger[14] says, "Most of the chronic sinus infections of adults can thus be traced back to early childhood."

Therapy is usually bed rest and cleansing of the alimentary tract. We rarely use local nasal medication although it is widely recommended. Dean and Armstrong[15] found that 80 per cent of their cases showed continued symptomatic relief after tonsillectomy and adenoidectomy.

Constipation is the favorite scapegoat of proponents of the toxemia theory throughout the nation. From a physiological standpoint constipation symptom-complexes are not the result of toxin absorption, but rather of pressure or blockage in the lower bowel. Best and Taylor[16] cite experiments proving this statement. Packing of the lower bowel with cotton, or inflation of a rubber balloon in the same region, gives almost instantly practically every symptom of constipation and its so-called "toxemia." William Baldwin, Professor of Physiology at the Philadelphia College of Osteopathy, recently quoted experimental physiological work wherein injection of the so-called toxic products of colon putrefaction in much larger doses than ever obtained from the human system failed to produce the clinical picture of toxemia from constipation.

This apparently disproves what is so generally accepted as the clinical picture of constipation seen in general practice. Whether true toxemia occurs, or whether Alvarez's explanation of mild reverse peristalsis is the answer, we do obtain clinical improvement by the elimination of constipation. We insist on at least two bowel movements a day. Clinical results seem to justify this arbitrary stand.

Urinary infections are one of the most common conditions of childhood despite the relative infrequency with which they are detected. The subacute and chronic infections are likely to pass unnoticed due to their mild symptom-complexes, until sufficient damage to body organs, especially the kidneys, has occurred to bring them to attention. Hematogenous, lymphohematogenous, and urogenous routes must be considered. Hematogenous infection is probably the most frequent. Campbell[17] suggests that we designate the condition as an acute or chronic pyuria until anatomic diagnosis can properly be made. "The usual lesion is a suppurative pyelyonephritis."[18] Campbell[19] offers us an excellent clinical corollary: "Do not fail to examine the urine of every child sick for any cause." We must remember, however, that "bacteriuria may be practically apyuric."[20]

Chronic urinary infections are best handled, at first, from a conservative viewpoint. We carry out the following regime:

1. Bed rest until the temperature has been normal for 48 hours. Recurrence of fever upon release requires reconfinement in bed.

2. High fluid intake is prescribed. One half-pint of fluid every hour on the hour, while the child is awake, is urged.

3. High caloric diet may be obtained coincident with the fluid intake by using fruit juices other than orange juice. Dextrose added to lemon juice and diluted with water can, by judicious mixing, produce a palatable and valuable high caloric, high vitamin C fluid.

4. Vitamin C either in 100 mg. tablets, or added to the above mixture as a powder, aids the body by replacing that lost during fever and during chemotherapy, if it is instituted.

5. An enema twice daily at first, in conjunction with a cleansing evacuant, such as castor oil, reduced to once per day as conditions warrant, is insisted upon. (These measures enumerated may be instituted immediately by the mother on telephoned instructions in a suspected urinary infection while awaiting the physician's visit.)

6. Osteopathic manipulative treatment, general in nature, to reduce spastic musculature and resultant abdominal somaticovisceral reflexes, to aid in lymphatic drainage, and to maintain the physiological state of all vital organs, is carried out as indicated in the individual patient.

7. Chemotherapy is not instituted until the alimentary tract is attended to, pH normalized and fluid balance stabilized. Sulfadiazine, mandelic acid and neoarsphenamine are being used to combat the microorganisms involved. If these measures fail after a four-week period, excretory urography is recommended. Instrumental or urosurgical treatment is required

for the urinary stasis usually found. "In one in approximately every 60 children with chronic pyuria the final diagnosis will be chronic renal tuberculosis."[21]

Undulant fever, a commonly overlooked infection which in chronic form smolders for years, simulating, like syphilis, almost any symptom-complex, must be considered. What child that visits the country, or travels through it, has not drunk unpasteurized milk? That is its most common source in this country. We must not overlook undulant fever because of its own pathological significance and its debilitating effects on the general resistance of the human organism.

We conclude that the perfectly healthy body, the one in which all organs, including the skin, are functioning physiologically, is capable of resisting all infection. It is only when either the external barrier —the integument—is broken, or the homeostatic powers inherent within the body are depleted, that infection occurs. Lowering resistance, in inverse ratio to virulence and numbers of microorganisms may result in the various symptom-complexes that today we call "infectious diseases."

Jordan and Burrows[22] tersely state the answer to our problem. "In general, resistance is at its height when the organism is functioning normally in every respect and is reduced by a variety of factors which interfere with and alter the normal physiological state."

Does this not sound similar to the following: "When you have adjusted the human body to the degree of absolute perfection, all parts in place, none excepted, then perfect health is your answer."[23] Resistance of the body is dependent upon normal functioning of all the body's parts. Normal function of the body is physiology. As Martin H. Fischer,[24] Professor of Physiology at the University of Cincinnati, has said: "Better learn physiology! For when the preventive medicine boys have conquered, only physiology will be left."

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Cervical Erosions*

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The uterine cervix is probably the most neglected portion of the female reproductive system in the routine examination made by the general practitioner. There is really no excuse for this neglect from the point of view of both health and actual danger to life. We pay a great deal of attention to foci of infection in the oral cavity. Why neglect the cervix, which is probably a more frequent offender and just as accessible for examination?

Owing to the strategic position and rich vascular and lymphatic supply of the cervix, it is in a state of turmoil throughout the greater part of a woman's life. Located between the germ-infested vagina and the sterile endometrium and peritoneum, it is the first line of defense against bacterial invasion. Endocervicitis and cervicitis are distinct clinical entities, separated from diseases of the uterus, tubes, and ovaries.

The structure of the cervix lends itself readily to infection, mainly owing to its close proximity to the anus and to the introduction of foreign bodies into the vagina. Instrumentation, coitus, and pregnancy add to the hazards. The deeply situated glands of the cervix make an ideal site for the harboring of an infective process.

The important role played by the cervix as a focus of infection capable of disseminating microorganisms and their toxins to more distinct structures is not fully appreciated by most practitioners. An infected cervix is the potent factor in the causation of tubo-ovarian disease, pelvic cellulitis, lymphangitis, and adenitis, the symptoms of which are pain in the lower abdomen and bladder, menstrual disorders, fatigue, headache, backache and general illness in addition to the usually recognized symptoms of cervical infection, such as leukorrhea and erratic bleeding. I believe the uterine cervix is the most important factor in pelvic disease and most resistant to satisfactory treatment.

The incidence of cervical infection is very large. When we consider that 78 per cent of all females between the ages of 20 and 40 years suffer from some form of this disease, we are made to realize the importance of it. This high percentage is directly or indirectly the result of bearing children. Childbirth may injure the cervical tissues, open up pathways of infection, and thus leave unhealed areas that may remain permanently unless corrected. Obstetrical injury, infection, or surgical operations which are not performed properly may produce a stenosis of the cervix, damming up infectious materials and producing serious trouble. These are the most common factors that make the cervix one of the most frequent sites of infection and chronic irritation in women.

The routine examination of all patients should start with a carefully elicited history. This not only provides information which is a great aid in arriving at a diagnosis, but at the same time gains the confidence and cooperation of the patient.

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In the examination of a female patient, special attention should be paid to the menstrual history, obstetrical history, and history of the present illness. Specific questions should be asked as to pain, its location, character, duration, relation to menstruation, associated symptoms and how relief is obtained. One should question as to abnormal bleeding; it is important to determine whether or not it occurs between menses or only at the time of flow, the color of the blood, presence of clots, and associated symptoms such as pain and secondary anemia. If leukorrhea is present, it is important to note its duration, the character, the color, and its relation to menstruation.

Pelvic Examination.—The cervix is so located that it is easily accessible for examination. Palpation is ordinarily carried out first. The examining finger determines its position, shape, consistency, eversions, size, lacerations, movability, and the presence or absence of fibroids and polyps.

No pelvic examination is complete without careful inspection of the cervix. The most common disorders are: Cervicitis and endocervicitis, erosions, cysts polyps, lacerations, and cancer. Special diagnostic procedures have been devised to aid in the differential diagnosis of cervical lesions, namely: Schiller test, colposcopy, and biopsy.

The Schiller test is simple to apply and is of value in routine examinations of the cervix. The procedure consists of painting the cervix with Lugol's solution. Normal tissues assume a mahogany-brown stain in about thirty sceonds, whereas diseased or suspicious tissues remain unstained and are white in color. These white areas may be due to: Trauma—fingers, speculum or instrument—leukoplakia, hyperkeratosis, erosions, Nabothian cysts. This test is to be used only when the cervix appears normal to the naked eye. Although it is not 100 per cent dependable, I believe it is an excellent office procedure and will oftentimes make us suspicious of a cervix that otherwise would appear normal.

The colposcope is of help only to the specialist who has had wide experience in its use, and I do not believe it practical for office use. It consists of a pair of binocular lenses which give an enlarged stereoscopic picture of the area examined. The prime purpose of the colposcope is to detect early cancer of the cervix. There is an early stage of carcinoma known as carcinomatous surface coating which may be detected with the use of the colposcope.

Although the Schiller test and colposcopy are helpful in establishing a diagnosis, the final decision must rest with microscopic examination of the tissue removed. The tissue may be obtained with a knife, or punch forceps, or with an endothermy knife. A fairly generous rectangular bit of tissue is taken from the area under suspicion.

Acute Cervicitis and Endocervicitis.—Inflammation of the cervix uteri may be either acute or chronic. Acute cervicitis is always of bacterial origin, usually the result of gonorrheal infection. Leukorrhea is the predominating symptom and the one that usually brings the patient to the doctor. In the acute stage this is purulent, and in the gonorrheal type there is vaginal and urinary irritability.

Diagnosis is made by inspection of the cervix. The cervix is congested and swollen. Yellowish purulent discharge is present. Smears from the cervical canal demonstrate the gram-negative diplococci.

Acute cervicitis and endocervicitis, irrespective of etiology, should not be treated actively, due to obvious risk of spreading infection. Such measures as local applications or cauterization are strictly contraindicated. Douches and vaginal tampons are not advisable. General hygienic care, such as rest in bed, light diet, care of bowels and ingestion of large quantities of fluid are helpful. The two most effective methods of treatment during the acute stage of cervicitis are the administration of sulfathiazole and the application of diathermy. Sulfathiazole appears to be less toxic than sulfanilamide or sulfapyridine. Dosage is 4 grams for the first day, then 1 or 2 grams daily for ten days.

After six to eight weeks, local treatment is usually indicated. Topical applications are of little value because of mucoid discharge. Cauterization of the canal with electric cautery, however, establishes free drainage, opens infected crypts, and destroys some cervical mucosa. One should be careful to avoid destroying all of the endocervical mucosa because of the danger of cervical strictures. There is an increased discharge for three weeks following which improvement begins and is progressive over a period of many months. Periodic examination with tests of the patency of the cervical canal should be made and re-cauterization should not be done within a year.

Diathermy has proved to be of value in the treatment of acute cervicitis. It is particularly advisable in cases of gonorrheal infections because it not only produces a cure in mild cases, but also in the more severe cases it actually brings about the disappearance or reduction in size of pelvic masses when these are present. The application of diathermy quickly relieves the abdominal and pelvic pain due to infection.

The effect on diseased structures is to produce an active hyperemia, the intensity of which depends on the degree of heat and the length of time the heat is applied. A quickening of the circulation and a dilatation of the capillaries occur. This produces a reduction of the stasis in the engorged blood vessels and relief of pressure on the nerve endings thereby decreasing pain.

Chronic Cervicitis.—This is probably the most common of all cervical lesions. While it may represent the residual phase of gonorrheal infection, it is more frequently due to infection by other organisms found in the lower genital tract, namely the streptococci, staphylococci and B. coli. In a large proportion of cases these organisms secure their lodgement as a result of childbirth trauma, instrumental injury, chemical irritation or physical irritation as from the use of a pessary.

The most common symptom is persistent leukorrhea. Other symptoms are low-back pain (often diagnosed sacroiliac strain), bearing-down feeling in the pelvis, dull pain in the lower part of the abdomen and urinary disturbances. In some cases there are dysmenorrhea and menorrhagia. I believe a badly infected cervix is a causative factor in arthritis and many times the cause of sterility.

Chronic cervicitis may present a variety of appearances. The irritation may be limited to one part of the cervix or it may cover the entire vaginal portion. The inflammatory lesion may follow an unhealed scar, presenting a red line of chronic irritation or granulation tissue. The cervix may be completely distorted by deep lacerations, may be swollen, edematous

or enlarged. The type of discharge varies considerably, but it is usually of a mucopurulent character.

The large hypertrophic cervix with its reddened, granular, and vascular mucous membrane may produce a picture simulating cancer. In any case a biopsy is indicated before instituting treatment.

The treatment of chronic cervicitis and endocervicitis is both preventive and curative. Prevention of gonorrhea will eliminate a large proportion of cases and proper obstetrical care will prevent an additional extensive group. Prophylactic measures during labor consist of waiting for spontaneous complete dilatation of cervix before attempting delivery, and the immediate repair of all cervical lacerations. Despite all the foregoing prophylactic care, more than half of all women who have had children have some degree of chronic cervicitis and must be treated accordingly.

Depending upon the nature of the cervical irritation, the treatment may be nonoperative, cauterization, trachelorrhaphy or cervical amputation.

Nonoperative treatment consists of:

(1) Application of diathermy.

(2) Normalizing the pH of the vagina and cervical canal. The normal pH of the vagina being alkaline and that of the cervical canal acid, any disturbance will lower the tissue resistance and encourage infection. This is the only condition in which alkaline or acid douches or powders have any value. The usual tampon and topical applications are of little practical value and I have discontinued their use.

(3) Copper Ionization: In the treatment of cervical infections, I believe ionization to be far superior to other forms of therapy. By ionization is meant the throwing off of the ions of certain metals into the surrounding tissues and these ions can be liberated only by a galvanic current. Some are thrown off at the positive pole and some at the negative. I use the copper electrodes. Copper ions stain the tissues green. The ions penetrate deep into the cervical crypts, thus eliminating infection most thoroughly with the least mutilation and alteration of structure and function.

Most backaches in women are due to, or aggravated by, infection of the cervix, which is carried through the lymphatics to the glands deep in the pelvis. These infected glands are tender, and if they lie close to the sacroiliac joints, they set up a chain of symptoms that is frequently referred to as "sacroiliac strain." The prompt relief afforded these patients is striking.

A galvanic current is used. The technic is as follows: A large dispersing pad, soaked in salt solution is placed on the lower abdomen and connected to the negative pole. The cord is attached to the positive pole, which is the intracervical electrode. The electrode should fit the canal snugly. The current is turned on slowly using 15 to 20 milliamperes for fifteen minutes. This treatment may be repeated weekly until a complete recovery is obtained.

(4) Colloidal Silver Intracervical Treatment: This technic is used in early cases of erosion where the infection is not extensive. The treatment differs from all other local applications in that it is not simply a surface treatment. A deep submucous penetration of the colloidal silver particles is obtained which results in stimulation of the reticulo-endothelial system, and hence maintains bacteriostasis and bacteriolysis. The method is so simple, safe, and painless, that it can be carried out by any family physician with good results.

The necessary equipment for this treatment is:

1. Graves' speculum
2. Plain probe for the introduction of the intracervical tampon
3. A specially prepared stringed tampon that is to be rolled on the applicator
4. Uterine dressing forceps
5. Common vaginal tampon
6. 10 per cent solution of argyrol
7. Argyrol glycerine solution
8. Glass slides for usual smear before treatment

The successful introduction of the tampon into the cervical canal requires that the position of the uterus and direction of the cervical canal be determined. After exposing the cervix, the vagina is cleaned with dry cotton balls and a smear is taken. No disinfectant of any kind should be used because of its possible reaction with sensitive colloidal solutions.

Success with this procedure requires careful selection and preparation of tampons. The intracervical tampon, made of the best long-fiber cotton and fibers running lengthwise, is wound smoothly on the applicator, dipped in the 10 per cent solution of argyrol, gently tucked in between the external and internal os, not higher. The argyrol solution should be fresh, not over three weeks old. After the introduction of the cervical tampon, a large retentive vaginal tampon, with a large knot on the end of the string that holds it together, is dipped in a 20 per cent argyrol glycerin solution and placed snugly against the cervix.

The tampons are left in place for from 20 to 24 hours and removed by the patient. Instructions are given to the patient to pull the string with the knot on first, and then remove the cervical tampon. This procedure may be repeated weekly until the desired results are obtained.

(5) Cauterization: One of the simplest and most successful office procedures, when properly carried out, is cauterization. Treatment by this means is painless; it is simple, inexpensive, and will cure most cases of chronic cervicitis, endocervicitis, erosions and Nabothian cysts.

The extent of the cauterization depends upon the nature of the lesion. A relatively superficial area of irritation is cauterized by a few light radial strokes. A deeper zone is destroyed by deep radial strokes to a depth of one-sixteenth of an inch the entire length of the eroded area, care being taken not to extend up too far into the cervical canal. This precaution must be observed to prevent stricture. If most of the cervix is involved, two or three incisions 1 cm. apart are made on the anterior and posterior lips and one incision at either angle of the cervix.

The postoperative care of the cauterized cervix is important. The cauterized tissue sloughs off in approximately ten days. Until it heals there is usually a profuse discharge lasting several weeks. The cervix should be inspected weekly and treated with some antiseptic solution to promote healing. Daily douches are advisable when slough is profuse. The patient should be instructed to return for periodic examinations to be sure that the cervical os is not constricted by scar.

Complications: About the only serious complication is hemorrhage. This usually occurs during the second week at the time the slough separates. It can usually be controlled by packing astringents, or the

cautery. Severe lesions will have to be sutured, which can be done without anesthesia.

Cervical Amputation and Trachelorrhaphy do not fall in the realm of the general practitioner. Nevertheless, there are instances in which these operations are indicated and should be referred to the surgeon. This is especially true if the cervix is greatly hypertrophied or so deeply torn that a cauterization cannot correct it satisfactorily.

Erosion is a complication of endocervicitis. It may supplement and increase a mucous endocervical discharge and the cervix may ooze slightly when traumatized. Upon inspection, the erosion is distinguished as a fiery red symmetrical lesion of the external cervix, centered about the external os and composed of granulations and endocervical mucosa. Erosions may be eradicated by ionization, cauterization or, in extensive cases, proper surgical care.

Nabothian or retention cysts of the cervical glands are benign and oftentimes present in conjunction with chronic cervicitis. Upon inspection they are translucent when they contain a clear mucus, or they may appear as opaque whitish blebs when the content is a viscid pus. When cysts are present, they should be punctured with the hot cautery tip. As soon as the cyst wall is punctured, mucus escapes. The wall of the cyst is then destroyed with the cautery tip.

Cervical Polyps are most frequently seen in adult women and are usually benign in character. Their chief significance lies in the fact that they are among the most common cause of intermenstrual bleeding. The symptom of bleeding, especially since it often occurs in the same age period as carcinoma, always causes apprehension. Therefore, all polyps should be subjected to a histological study. The treatment is removal. This is best done by cautery at which time the base is destroyed to prevent recurrence.

Carcinoma of the Cervix Uteri is by far the most important of all cervical lesions. When we consider that the cervix is the seat of 90 per cent of all uterine cancers, there is no excuse for any physician who does not have it constantly in mind. Although the treatment of cancer is not in the realm of the general practitioner, the unearthing and diagnosis of early cancers of the cervix are his responsibility and in the end will save many lives.

A carefully elicited history, which is so often neglected, will prove to be of great value. Although it is impossible to make a diagnosis on clinical symptoms alone, the value of such symptoms will make one think of the possibility of cervical cancer.

A history of abnormal bleeding or discharge in women past thirty should always be considered indicative of cancer until proved otherwise. Bleeding is usually the first symptom of cancer of the cervix. During the menstruating years of life, intermenstrual or contact bleeding, even though very slight, should arouse suspicion. It is apt to be noted after exertion, coitus or the straining of defecation. The contact bleeding following coitus or special pelvic examination is characteristic.

Abnormal discharge, usually watery in character, may be present before the appearance of bleeding. However, this discharge later becomes tinged with blood and as the disease progresses, the bleeding and discharge become more profuse, and secondary infection produces an offensive odor.

Pain is not a symptom of early cervical carcinoma, and is usually not present until the late stage of the disease.

Pelvic examination in suspicious cases should consist of both careful palpation and inspection. Palpation may reveal the presence of a malignant growth. Most frequent location of early cervical cancer is a small lesion at or near the external os. To the palpating hand it appears as a small hardened area often slightly raised above the surrounding tissues.

Careful routine inspection of the cervix is probably the greatest aid in the early recognition of cancer of the cervix. It appears as a granular area, slightly elevated, and bleeds on slight touch, as from contact of speculum or examining finger. The surrounding cervix may be normal or the seat of chronic inflammatory disease.

Of the special diagnostic methods used, I believe the Schiller test, although not diagnostic in itself, may prove of value to the general practitioner. It will at least make him suspicious of cervical lesions and probably make him more cancer conscious.

For final diagnosis, the biopsy or microscopic tissue study is most important. Any suspicious area of the cervix should be considered cancer until tissue study proves otherwise. If this simple rule is followed, many early cancers of the cervix will be uncovered, thus avoiding unnecessary suffering; what is more important, life will be preserved. Once the diagnosis of cancer of the cervix is made, proper treatment should be instituted. This is not an office procedure, but belongs to the trained gynecological surgeon.

CONCLUSION

It is clear that the cervix presents a wide variety of proliferative lesions. These are present in old chronic irritations, infections and childbirth injuries. The vast majority of these lesions are benign, a few are definitely malignant, and occasionally one finds in these chronic irritations an early, noninvasive carcinoma. It is in these chronic irritations that practically all noninvasive carcinomas have been found. To the naked eye, they all look like chronic unhealed lacerations, eversions, and areas of irritation. We do not know what changes these benign, inflammatory tissues to malignancy, but we do know that inflammatory lesions are unhealthy and are the soil in which carcinoma has grown.

Therefore, the responsibility of the general practitioner is as follows:

(1) Emphasis on the value of periodic pelvic examinations
(2) Carefully elicited history
(3) Routine pelvic examination—both palpation and inspection
(4) Early recognition of cervical cancer at a stage when proper treatment can produce a cure
(5) The use of special diagnostic methods
(6) Microscopic study or biopsy of all suspicious tissues
(7) Correction of conditions of chronic cervical irritation, which may be predisposing factors.

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Malaria: Diagnosis and Treatment*

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Malaria will be a problem in the near future for many of us who have never seen much, if any, malaria in the past. The average individual upon hearing the word "malaria" immediately thinks in terms of mosquitoes. Let us, however, confine our interest to an individual with an obscure fever of malarial origin.

The word "obscure" means difficult for the eye to see and the mind to discern. To help our eyes to see and our minds to discern let us present a verbal picture of an individual with the perverted physiology caused by an invasion of malarial parasites.

It is variously estimated that there must be a minimum of 500 to 1500 parasites to the cubic millimeter of blood to cause fever. This is designated by Ross as the pyretogenous limit. According to various authorities the fever is caused by the rupture of parasitized erythrocytes, with the release of debris, cell fragments, products of the parasites and the parasites themselves into the blood plasma. Abrami and Senevet regard the malarial paroxysm as being at least partly an anaphylactic process. When the parasites are again within the erythrocytes and undergoing the growth process, the toxic effects are not felt; therefore, the temperature returns to normal until the next segmentation occurs.

At this time I would like to point out that from no matter what cause, the rapid destruction of blood is associated with aching pain in the back, abdomen, or limbs, headaches, malaise and a severe, shaking chill. The individual then has a fever, caused either by the anaphylactic shock of the outpouring of parasites into the blood plasma, or by the rapid destruction of blood, or by both.

In the opinion of the writer the variety of symptoms is caused as much by the difference in makeup of individuals as by the difference in the types of plasmodia. We need to consider that any involvement of the erythron of necessity is reflected in the physiological response of the entire hematopoietic system.

This fever is characterized by a periodicity and intermittency paralleling the maturation of the parasite, this maturation producing wholesale hemolysis.

We have, then, an individual with a regularly intermittent fever, suffering from a hemolytic anemia. The severity of the anemia is in large measure directly correlated with the intensity and duration of the infection. Normally about one trillion, or one twenty-fifth of all the circulating erythrocytes, are disposed of by hemolysis each day. An old cell, or an odd-shaped or odd-sized cell, begins fragmentation and is taken out of the circulation by the reticulo-endothelial cells, principally in the spleen. The hemoglobin within the interstices of the stroma does not return to the blood but is disintegrated by the reticulo-endothelial cells, the globulin-hematin fraction is split off, and bilirubin is formed.

The spleen, being the principal organ of blood destruction, must of necessity enlarge its capacity in the event of an increased number of abnormal red cells, such as the swollen cells which have been invaded by malarial parasites.

The individual under consideration has an intermittent fever coming at regular intervals, a hemolytic type of anemia, and an enlarged spleen.

The liver responds to this excessive amount of free hemoglobin by an increased secretion of bile. There is a resulting congestion in the liver, and enlargement usually accompanied by hepatitis. The increased secretion of bile, especially if unmodified by the action of hydrochloric acid, causes vomiting and diarrhea similar to that seen when a person takes too much bile salts as a purge. The writer has noted this particularly in individuals where there was little or no free hydrochloric acid in the stomach. We must keep this in mind when treating the individual and instead of treating our bilious patient with calomel, we should give him dilute hydrochloric acid with his meals. This has also been noted by the writer in treating cases of pellagra and milder cases of vitamin B deficiency where diarrhea has been a prominent symptom.

The individual usually has an increased icterus index, due to increasing amounts of bilirubin and other pigments—the result of excessive blood destruction. This will vary all the way from a slight rise of the icterus index to a frank clinical jaundice, discoloring the skin and the whites of the eyes.

The tongue appears to be of a doughy consistency, and upon protrusion it will be noted that the teeth have left their imprint. I do not know that this is characteristic of malaria, because I have seen it in other debilitated states. However, I have never seen a case of malaria existing for some time in which this was not present.

Clinically, then, we have an individual with fever which is usually preceded by the chill, followed in an hour or two by a drenching sweat, of peculiar odor, an enlarged spleen, a swollen, tender liver, and a progressive anemia, hemolytic in type.

Malaria, however, can no more develop according to a simple pathological pattern than can tuberculosis, syphilis, or any other disease with a variable clinical course. The duration, severity and outcome are affected by a number of variables, such as the species of the parasite, the variation in its biologic properties, the length of infection, the age and race of the host, the differing grades and kinds of resistance, the effects of relapses, the kinds and amounts of therapy used and the concomitant influences of intercurrent and terminal infections.

The type of fever caused by the *Plasmodium vivax*, or tertian malaria, and that caused by *Plasmodium malariae*, or quartan malaria, are similar clini-

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cally except that in the former the paroxysms occur at 48 hour intervals and the latter at 72 hour intervals, the time required for the parasite to undergo schizogony.

Clinically these types consist of three well-defined stages: (1) The chill or cold stage, (2) the hot or fever stage, and (3) the sweating stage. They are clear cut, easy to recognize and amenable to treatment.

Let us, therefore, concentrate on the clinically important estivoautumnal fever which more often terminates fatally and because of its protean character is likely to be confused with other diseases such as typhoid fever, bacterial septicemia and cerebral disturbances caused by other conditions. It differs from the other two types of malaria in the following respects: The rigor is not so pronounced and the fever is very irregular, extending over a much more prolonged period. Often severe headache, persistent vomiting, diarrhea and hyperpyrexia accompany these stages. At any stage of the infection more pernicious characteristics may develop. Often the patient is comatose before he receives medical attention. Destruction of red blood cells is particularly marked in this infection. If the patient survives long enough, there develops great pallor of mucous membranes and conjunctiva, retinal hemorrhages, cardiac distress and dyspnea, erythrocytes under 1,000,000 to the cu. mm. of blood, extreme leukopenia, hemoglobin 10 per cent, microcytes and megaloblasts in the peripheral blood. The main symptoms may be of the central nervous system, involving various nerve centers. A hyperpyrexial temperature as high as 112 F. may develop. There may be convulsive, epileptic or tetanic, and paretic symptoms simulating those found in meningitis, insanity, etc. There is the algid or cold sweat variety, and the choleraic and dysenteric forms characterized by numerous loose stools, great loss of fluids and maybe hemorrhage from the bowel.

To summarize malaria clinically, no matter which type of plasmodia is the offender, we have constantly, although in varying degrees, the following:

(1) Fever, preceded by chill and followed by sweating. This fever is *(a)* intermittent, *(b)* periodic; (2) anemia, (3) enlarged spleen, (4) enlarged tender liver, and (5) yellow discoloration of sclera and skin.

The definite diagnosis depends entirely on the laboratory findings which are:

(1) Erythropenia, progressive diminution in number of red blood cells, down as low as 1,000,000

(2) Progressive lowering of hemoglobin paralleling the lowering of the red blood cell count

(3) Red cells normal to small in size

(4) Total white count varying between 5,000 and 10,000

(5) Differential count in the chronic variety reveals relative monocytosis. In the acute variety, an increase in stab cells

(6) In the study of whole blood *(a)* the sedimentation rate is increased, *(b)* icterus index is increased according to the degree and rapidity of the blood destruction; *(c)* hemotocrit reading lowers proportionately with the anemia.

(7) Hemoglobinuria, bilirubinuria

(8) The Kahn or Wassermann tests will be positive in about 25 per cent of the cases of malaria, even though the individual is not syphilitic. They usually become negative after 10 days of intensive malarial therapy.

No definite diagnosis can be made of malaria unless the parasite can be demonstrated in the red blood cells. This can be done by various methods.

I will give you verbatim the method used by the Bureau of Laboratories of the Florida State Board of Health.

PREPARATION AND STAINING OF FILMS

Use only clean, preferably new, slides. Used slides are permissible, provided they are free from scratches and corrosion.

Thick Films.—

(1) A large drop of blood is spread on one end of slide over an area of one-half inch in diameter with a needle or toothpick or by tilting the slide

(2) Dry thoroughly in the air

(3) Number slides with wax pencil on opposite side from smear

(4) Cover smear with dilute Giemsa stain (1 drop to 1 cc buffered distilled water having a pH of 7.0 to 7.2) for one hour

(5) Wash gently and dry thoroughly in the air

(6) Examine with oil immersion lens

Thin Films.—

(1) A thin film is frequently necessary for specific identification of the type of parasites and should be made on one end of the slide containing the thick film or on another slide

(2) Dry thoroughly in the air

(3) Number slide with a wax pencil on the opposite side from the smear

(4) Fix the smear with **methyl alcohol**

(5) Cover smear with dilute Giemsa stain and allow to stand forty-five minutes

(6) Rinse in running water and allow to dry in the air

The method used by the Florida Bureau of Laboratories is rather time-consuming, and I suggest for those doing a considerable amount of this work the method of Fields or the modification of it as used by the Army in the Pacific. The fundamental theory of this method was reported by Simons in 1938 and Pampani in 1938 namely: That the hemoglobin is used to provide a color contrast, or in effect, to act as a counterstain. The stained parasites and the leukocytes are contrasted against a background of laked red blood cells.

In making a "thick-thin" smear, take a clean or preferably new clean slide, and with a wax crayon draw a heavy mark across the slide about three-quarters of an inch from one end. Cleanse a finger dry and prick with a lancet. A drop of blood about the size of a small pea is placed on the smaller area of the slide. Utilizing the ball of the bleeding finger as a spreader, the drop is spread to the size of a five cent piece and thin enough to see the hands of a watch through the film.

A very thin film is spread at the opposite end of the slide.

With the thick portion up, the slide is placed in a semi-vertical position to dry and the excess blood from the thick smear is allowed to drain to the crayon mark. The slide is air dried in this position and protected by a screen if there are flies about. The thick smear is ready for staining as soon as it appears dry.

The film is dipped for 1 second in Sol. A. It is immediately dipped in clean water for a few seconds or until the stain ceases to flow from the slide. It is then dipped in Sol. B for 1 second. The stain is flushed off and then rinsed gently in clean water. It is dried in a vertical position.

To make Fields' stain,

MODIFIED SOLUTION

1.25 grams of methylene blue chloride and 5 grams of sodium phosphate dibasic (Merck) are dissolved in 50 cc. distilled water. Bring to a boil and evaporate to dryness. Then add 6.25 grams potassium phosphate monobasic (Merck) which is dissolved in 500 cc. of distilled water. Mix well and let stand for a few days. Filter before using.

SOLUTION B

Dissolve 5 grams of sodium phosphate dibasic (Merck) and 6.25 grams potassium phosphate (Merck) in 500 cc. distilled water. Add Eosin Y 1 gram. This solution must be replaced when it becomes greenish.

After the thick portion of the smear is stained and dried, the thin smear is now ready for staining. A combination of Wright's and Giemsa's stains gives nearly perfect results.

Fourteen to 16 drops of Wright's stain are floated in the center of the film. This is allowed to stand for 30 to 60 seconds to fix the stain. Then it is diluted with 24 to 26 drops of distilled water. The mixture is allowed to stand for from 8 to 12 minutes. The slide is washed well with water and then is decolorized with 95 per cent ethyl alcohol until it is a pale pink color. Freshly filtered stock Giemsa stain is diluted with 15 parts of distilled water and placed in a petri dish. The thin portion of the smear is now placed face down in contact with the stain in the petri dish for 20 to 30 minutes. It is then washed and dried.

The appearance of the malarial parasites in the thick smear is very different from that in the thin smear. Whereas in the thin smear the parasite with its typical red-staining chromatin dot and blue cytoplasm is found in the infected red blood cell, in the thick smear the red blood cells are destroyed leaving the parasites free and concentrated. Under oil immersion the thick smear is examined at the junction of the circumscribed portion and the finger-like projection where the blood has drained toward the center of the slide. This portion is clearly seen when the slide is placed on a white background. It appears as a thin wavy brown-red line. This is not only the area of maximum dehemoglobinization, but it is at this point that the parasites tend to cling when the blood is dried in a semi-vertical position.

In a smear which contains parasites and which is stained by Fields' method, the chromatin dots stain a bright red to a crimson and can easily be seen. In many instances the blue cytoplasm is found coexistant with the chromatin dots, confirming the impression that plasmodial parasites are present in the smear.

In the thick smear parasites appear smaller and more shrunken than in the thin smear. The thick smear is used to confirm the presence of malarial parasites in the blood. The thin smear is used to differentiate the type of plasmodia present.

This is important in treatment. In a case of benign tertian malaria, with little or no fever and where only a solitary parasite is found in the peripheral blood, nothing has been lost if one has simply awaited developments. However, in subtertian malaria the treatment should begin as soon as the diagnosis has been made. Delay in treatment in estivoautumnal malaria means a lost opportunity, for there is always risk of serious development at any time. In fact this is the reason of prime importance for trying to classify the type of plasmodia present. It is also most important in prognosis.

In the blood smear *Plasmodium vivax* is characterized by a single red chromatin dot, associated with blue cytoplasm. This has a tendency to assume many variations in form dependent on the stage of the parasite. This parasite enlarges the red cell, and makes it pale. Schueffner's punctations may be observed. The ring form is large, about one-third the diameter of the red blood cell. Number of nuclei in division forms is 12 to 22. The shape of the asexual form varies greatly; the mature forms are usually round or oval. The sexual forms are usually slightly larger than the red blood cells.

The *Plasmodium malariae* usually reduces the size of the red blood cells; it intensifies their color. There are no Schueffner's dots. The ring forms contain one nucleus and sometimes two. The division forms never contain more than 12. The asexual forms are usually bandshaped, while the mature forms are oval or round. The sexual forms are round or oval and smaller than the red blood cells. There is always present a yellow pigment in the asexual forms.

The *Plasmodium falciparum* does not affect the size nor the color of the red blood cell. The ring forms are very small (about one-fifth the diameter of the red blood cell) and always contain two or more nuclei. There are 8 to 12 to 32 nuclei in division forms. The asexual forms are not seen. The sexual forms are crescent-shaped and about one-half the diameter but slightly longer than a red blood cell. Occasionally there is a yellow pigment in asexual forms.

TREATMENT

General: The patient should be placed in bed at absolute rest. Evacuation of the bowels should be secured either by enema or cathartic. The patient should have symptomatic treatment, the head pain be relieved with aspirin, or if more difficult an Empirin Compound No. 3 every 3 to 6 hours. In the "cold" stage added blankets, hot water bottles, and hot lemonade to which whiskey has been added are indicated. Citrocarbonate drams 1, in a glass of water 6 to 8 times a day is beneficial. This is especially important in what is known as blackwater fever or hemoglobinuria. It prevents the formation of acid hematin which may become great enough to plug the tubules of the kidney causing anuria, the retention of nitrogenous products in the blood, and death. The mortality rate of blackwater fever is about 25 per cent and we must be on the lookout for it in malaria, especially in the estivoautumnal type.

We must not forget to alkalinize these patients immediately, to prevent the formation of acid hematin in the tubules of the kidney.

The diet is to be liquid or light.

Malaria complicated by pregnancy is given the same treatment. Quinine doesn't produce labor pains but it will make them stronger if they are present. Malaria, however, may cause abortion and delivery may cause a relapse of a successfully treated case. The greatest cause of subinvolution in the South is in my opinion malaria.

The active treatment of malaria is medicinal! Quinine continues to rank high as an antimalarial agent. There are two schools of thought on its administration. The long, or standard, quinine treatment and the short quinine treatment. In this country quinine sulphate is usually given in either case. In the long treatment 30 grains are given in divided doses each day for three weeks and then 10 grains each night for 8 weeks.

The short treatment has several advantages and is the one I personally prefer. It is cheaper and less apt to produce symptoms of cinchonism. It is far easier for the patient to follow the physician's advice and it brings out the full tonic effect of the quinine because of the moderate amounts employed. I prefer giving quinine sulphate grains 5 four times a day after meals and at bedtime until the fever is gone and then continue this 4 or 5 days longer. In my experience relapse is no more common with the short than the long treatment.

Relapses (or reinfections) are to be treated exactly as the initial attack. The short treatment is not only effective in controlling the clinical symptoms but also it avoids the useless procedure of trying to destroy the few surviving parasites which may by their presence help build up some immunity in the individual. The short quinine treatment is much safer. There are very few who cannot take quinine—perhaps less than one-tenth of 1 per cent.

If because of pernicious vomiting or coma one cannot use the oral route, then quinine dihydrochloride must be given paraenterally—either intramuscularly or intravenously. It must be modified and can be obtained in sterile ampules. Sterile distilled water is added. The intramuscular route may cause considerable pain and even necrosis. Because of this I prefer the intravenous route. Never more than 8 grains should be given intravenously as it will lower the blood pressure and the patient may go into shock. When given intravenously it is repeated at 4 to 6 hour intervals until the patient can take quinine by mouth. In malaria we should always give medicine by mouth if at all possible.

Children generally tolerate quinine very well and the fraction of the adult dose is usually larger than in other drugs. Children under one year of age may be given 1 or 2 grains a day, usually .2 grain for each month of age. Older children are given 1.5 grains of quinine sulphate for every year of age until the adult dose is reached. This may be given in the form of pills, capsules or tablets. In case these cannot be swallowed (by young children for instance) administration can be accomplished with quinine tannate, given in honey, jam or chocolate. If oral administration is not possible, a very rapid effect can be secured without injections by suppositories of quinine and urea hydrochloride, which is extremely soluble and quickly absorbed.

Atabrine acts directly on the trophozoites and schizonts of all the species of plasmodia and upon the gametocytes of *Plasmodium vivax* and *Plasmodium malariae*, destroying them. It is supplied in tablets grains 1½ and the recommended dosage is one tablet three times a day for 5 days. If there is a yellowish discoloration of the skin and conjunctiva the drug should be discontinued. This is not due to its toxic effect but to the deposition of the dye. As the drug is cumulative even normally, it should be discontinued before toxic symptons develop, such as colic, nausea, vomiting, headache and nervousness.

The dosage of a grain and a half three times a day for 5 days should not be exceeded. The recommended daily dosage in children: up to one year, ¾ grain; from one through four years, 1½ grains; from five through eight years, 3 grains; over eight years the adult dose may be given. This drug may also be obtained in ampules for intravenous therapy in patients who cannot take anything by mouth.

Plasmochin should not be used in the treatment of malaria at all, but only to eliminate gametocytes. It should never be given within three days of the administration of atabrine as the two drugs seem to enhance the toxic effects of each other. The method of administration of Plasmochin is by mouth; it may be obtained in a tablet form either alone or in combination with quinine. The dose is relatively small, one-sixth grain three times a day for five or six days.

In closing I would like to mention that in this country in 1941 there were 1,178 certified deaths due to malaria. In 1933 there were 4,678. This is indicative of a tremendous loss of manpower as there are approximately 1,000 individuals incapacitated annually for each reported death. This is a challenge to us because the diagnosis is relatively easy and there are specific drugs available for the treatment.

119 Dakin Ave.

ANNUAL STUDENT ESSAY CONTEST

Students in osteopathic colleges for the past several years have had an opportunity annually to participate in an essay writing contest. For this year the subject is "The Value of Osteopathic Manipulation in Prenatal Care."

A cash prize of $25.00 will be awarded to the writer of the best paper submitted in each approved college of osteopathy in which there are four or more contestants. If at least four schools have a minimum of four contestants each, a grand prize of an additional $25.00 in cash will be awarded to the writer of the paper adjudged to be the best among the prize-winning papers from the competing schools.

The competition is to be judged on the basis of papers of not more than 2,000 words on the assigned subject.

It is the donor's wish that as many undergraduates as desire shall participate in the contest in each school. The main purpose of the contest is to promote original investigation and thought among osteopathic students and the contest will doubtless be more worthwhile if instructors in each of the colleges will direct the attention of the contestants along practical clinical research lines.

Instructions for preparing papers are posted in the various osteopathic colleges.

The winning manuscript from each college must be received in the Central office of the A.O.A. not later than April 15, 1945. The deadline for the receipt of manuscripts in each college will be set and announced by the committees in the respective colleges.

The winning manuscript in each school, properly identified, will be judged by a committee set up by the Executive Committee of the American Osteopathic Association. The announcement of the award will be made in the general sessions during the annual meeting of the Association in July, 1945, in Kansas City, Mo.

The donor reserves the right of publication of any or all of the winning essays.

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BOARD OF TRUSTEES MEETS

The Board of Trustees of the American Osteopathic Association met in Chicago December 7 to 10. In previous years only the Executive Committee had met at the midyear. Every member of the Board was present though some of them had considerable travel difficulties. The American Association of Osteopathic Colleges held its midyear meeting on a part of the same days.

The highlight of the meeting had nothing to do with business. Dr. Phil R. Russell, former president of the American Osteopathic Association, had a son in the air forces when Japan attacked the Philippine Islands. He escaped capture and commanded a troop of guerillas until orders came through to surrender. From that time he was a prisoner of the Japanese until September 7 when the ship on which the Japanese were moving prisoners was torpedoed and sunk by American forces. He was rescued and taken to New Guinea September 23. He arrived in San Francisco December 1; was married December 6 there and flew to Washington, stopping in Chicago to give his father the first glimpse he had had of his son since the war began. He was welcomed by the members of the Board.

Among the appointments made by the President and approved by the Board are the following additions to boards and committees:

Advisory Board for Osteopathic Specialists—Dr. Allan A. Eggleston.

Osteopathic Research Council—Drs. S. V. Robuck, Leonard V. Strong, Jr., W. Fraser Strachan, Paul T. Lloyd.

Committee on Professional Visual Education—Wallace P. Muir.

The date for the annual conference in Chicago of A.O.A. and divisional society officers, the first of which was held in February, 1944, was set for February 2, 3 and 4.

The Constitution and By-Laws of the American Osteopathic College of Radiology were approved.

In connection with the American Association of Osteopathic Colleges plans were discussed at length, and advanced, for the continuation of expansion programs and for the prosecution of fund-raising activities.

Plans were made for advanced vocational guidance and student selection work, including especially, the undergraduate and graduate education of men now in the armed forces.

The minimum standard curriculum for osteopathic colleges was revised, as well as the standards for approval of osteopathic colleges and for hospitals for intern training. Also a standard was set up for hospitals to be registered.

The procedure for the work done in the Division of Public and Professional Welfare was revised.

The hospitals approved for intern training were:

College Hospital, Los Angeles, Calif.
Corpus Christi Osteopathic Hospital, Corpus Christi, Texas
Green Cross Hospital, Akron, Ohio
Waterville Osteopathic Hospital, Waterville, Maine

This makes the complete list:

Amarillo Osteopathic Hospital, Amarillo, Tex.
Bangor Osteopathic Hospital, Bangor, Maine
Bashline-Rossman Hospital, Grove City, Pa.
Battle Creek Osteopathic Hospital, Battle Creek, Mich.
Carson City Hospital, Carson City, Mich.
Chicago Osteopathic Hospital, Chicago
Cleveland Osteopathic Hospital, Cleveland
Coats-Gafney Clinic and Hospital, Tyler, Tex.
College Hospital, Los Angeles, Calif.
Conley Clinical Hospital, Kansas City, Mo.
Corpus Christi Osteopathic Hospital, Corpus Christi, Tex.
Dayton Osteopathic Hospital, Dayton, Ohio
Des Moines General Hospital, Des Moines, Iowa
Detroit Osteopathic Hospital, Detroit
Doctors' Hospital, Inc., Los Angeles
Doctors' Hospital, Columbus, Ohio
Donovan Osteopathic Hospital, Raton, N. M.
Gleason Hospital, Larned, Kans.
Gorrell Hospital, Corpus Christi, Tex.
Green Cross Hospital, Akron, Ohio
Gribble Hospital, Vidor, Tex.
Hillside Hospital, San Diego, Calif.
Hustisford Hospital, Hustisford, Wis.
Joplin General Hospital, Joplin, Mo.
K.C.O.S. Hospital, Kirksville, Mo.
Lakeside Hospital, Kansas City, Mo.
Lamb Memorial Hospital, Denver
Lancaster Osteopathic Hospital, Lancaster, Pa.
Laughlin Hospital, Kirksville, Mo.
Los Angeles County Osteopathic Hospital, Los Angeles
Madison Street Hospital, Seattle, Wash.
Magnolia Hospital, Long Beach, Calif.
Massachusetts Osteopathic Hospital, Inc., Boston
McLaughlin Osteopathic Hospital, Lansing, Mich.
Mercy Hospital, St. Joseph, Mo.
Monte Sano Hospital and Sanitarium, Los Angeles
New Mexico Osteopathic Clinic and Hospital, Albuquerque, N. M.
Northeast Osteopathic Hospital, Kansas City, Mo.
Osteopathic Hospital of Maine, Inc., Portland, Me.
Osteopthic Hospital of Philadelphia, Philadelphia
Osteopathic Hospital of Rhode Island, Inc., Providence, R. I.
Ozark Osteopathic Hospital, Springfield, Mo.
Rocky Mountain Osteopathic Hospital, Denver
Saginaw Osteopathic Hospital, Saginaw, Mich.
Southwestern Osteopathic Sanitarium & Hospital, Wichita, Kans.

Sparks Clinic and Hospital, Dallas, Tex.
Stone Memorial Hospital, Carthage, Mo.
Tulsa Osteopathic Hospital, Tulsa, Okla.
Waldo General Hospital, Seattle, Wash.
Waterville Osteopathic Hospital, Waterville, Me.
Yakima Hospital, Yakima, Wash.

EDITORIAL POLICY—A RESTATEMENT

An editorial writer in the JOURNAL for September, 1944, mentioned the advantage to an osteopathic physician of being "fortified by the knowledge and experience of others, even though that experience may not be 'validated' by the explanations of science." One cannot too strongly emphasize the hope to which the JOURNAL frequently has given editorial expression that original osteopathic research will be prosecuted both by individuals and by institutions, and that the result will be published. It is our belief that every bit of work so reported serves as a stimulus, and often it is a guide, so that more will be done.

In that connection it may be well to reproduce certain paragraphs from an article on "Some Principles of Writing for A.O.A. Publications," which appeared in this JOURNAL in March, 1943. At that time the Editor said:

"The literature of the profession, in the form either of books or periodicals, has lagged . . . so that undue dependence has been placed upon that produced by other schools of healing . . .

"[We are] undertaking to stimulate interest on the part of foundations, and in philanthropically inclined individuals who, before they remember us in grants, or bequests, will have us investigated by the best talent they can secure. To this end we have brought in experienced men from the general field of education to study our institutions and make recommendations for their improvement. Our literature as well as other expressions of our activities must meet their scrutiny. It is natural to expect these people, well-informed and cautious as they are, to make their own investigations and also to make sure that their friends and perhaps their agents—trained men, educators, scientists, look at us from every angle. If it be that such an expert can place his finger upon crudities and absurdities in our journals, even though he be inclined to be fair to us, or more than fair, he can in all honesty do only one thing—and that is, blast us so far out of consideration that we may never be able to come back in the particular enterprise in question. . . .

"The rising quality of our own students, the higher educational standards which we demand of people even before we will invite them to consider entering our profession, make it essential that our literature be as good as we can make it. . . .

"The Executive Secretary and the Editor often have stressed . . . the importance of taking one small point in a subject and carrying it further than anybody ever had before. . . .

"There is a vast range of facts which have not been brought to general attention. There are innumerable studies which should be made, each starting with a known fact and going on out into the things as yet unknown. It is also true that certain studies have been made which need to be supplemented, or which need to be repeated for the purpose of finding how nearly the experience of other investigators coincides with those which are published. Each of these provides material for a useful and valuable article or articles. . . .

"Some time ago there was an editorial in a daily newspaper in which there was a reference to 'the mistake hound who looks at life as a series of bonehead plays on the part of somebody else and wants to exploit himself in exploiting them.' Although it sounds bad, yet perhaps something of the quality described there is absolutely essential in the make-up of an editor. Let us set over against that a few sentences clipped years ago from a review of what was then a new edition of 'Encyclopedia Britannica.'

'First of all one expects accurate and authoritative articles. This means that they must be substantially free from error in the light of contemporary knowledge, and be written by men of established reputation. Both requirements are extremely difficult to comply with. Accuracy of statement is a fine art for which exact knowledge is only the first essential . . .' . . .

"Why is such accuracy necessary? . . . We have been severely criticized about many things. Perhaps none of this criticism has been more bitter than in connection with our treatment of books written by osteopathic physicians. There was one particular instance wherein the Editor condemned a book because he considered it in general a disgrace to the profession. A remark like that, and other things sometimes said by the Editor, lead some in the profession (again not accurate thinkers) to come to only one conclusion: that the Editor constantly sets up the American Medical Association as an arbiter of our own policy and work. The Editor cares no more than anyone else in the profession what the American Medical Association may think of our standards. But our standards and our literature should be of such a nature as to stand up in the scientific world under the same scrutiny which theirs must meet. . . .

"If the Editor insisted upon putting nothing into THE JOURNAL unless he, of his own editorial knowledge, knew it to be true, it would fall pitifully short of what it ought to be. He must have confidence in the pioneering writers who are developing the things which can make a good periodical. . . .

"It is amazing to one who never has given the matter thought, to realize how much of what must be true in anatomy and physiology has never been found out, and how little there is which is known. It is amazing to know how much is simply assumed and accepted as fact, and passes from book to book, generation after generation, without any verification. There are many who do not understand that the Editor recognizes this state of affairs. They believe that he demands that everything presented for publication shall accord with the generally accepted beliefs. He does not. But he does demand that when it does not

accord with what he understands osteopathic or other scientific belief to be, some reason shall be given for the divergence, and that, usually, he has trouble in getting.

"Because of the Editor's attitude on this point it is often charged that he turns down the best of osteopathic material, and constantly demands that whatever is offered for publication must accord with published M.D. beliefs, so many of which, say his critics, have been discredited. This charge is not well founded. But it is true that when one presents a basic statement, he is expected to justify it either by the fact that it is already known to be true, or by stating wherein it varies from the generally accepted belief and why he thinks the accepted belief is wrong.

"When a contributor makes a statement which is new, or which has a new application, he is expected to substantiate it by showing either where its truth can be proved in osteopathic or other literature, or how, by experiment, observation, or logical reasoning, he learned it. He must never simply set it down and let it stand, bare and dogmatic.

"When a person presents an idea relating to the form or function or disease of some part of the body, he is presumed to have studied the standard books to be absolutely sure of the beliefs held by other people about the region under consideration. As he has studied and thought, he is presumed also to have kept in touch with current periodicals and books as they have dealt with the subject He is supposed to go into a discussion of those things and particularly what he has done personally toward demonstrating the truth of, and finding flaws in, his hypothesis. It is to be remembered that while Dr. Still struggled with his problem through the years he worked from at least two angles:

"(1) He constantly studied the living body and also the internal machinery of bodies which had been living

"(2) And there is no doubt about the anatomy texts which he wore out by constant study.

"It is a part of the preparation of osteopathic physicians that while in school they apply themselves to the study of books and to the study of living bodies and of cadavers, and at the same time, to the intensive training of their own perceptive powers. For instance, a student will put a hair in a textbook, turn one leaf over it, and pass his fingers over it until his tactile sense is keen enough to distinguish just where this hair is. Then he will palpate through two leaves of paper, and so on, constantly intensifying his tactile sense. When one works out a theory he likewise devotes days and nights to the task. It is not idle boasting for him, when he comes to write an article, to report the steps he took. Rather it is in order to report the study upon which he bases the beliefs he holds."

Those paragraphs, taken from an address originally delivered years ago, express the policy which has guided the editorial department of THE JOURNAL through the years.

SPECIAL ARTICLE

The Teaching of Biochemistry

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It is only necessary to be acquainted with the syllabi of the different courses in biochemistry as offered in various medical colleges or to examine the texts in this subject prepared for the use of medical students to be convinced that there is a considerable difference of opinion as to what constitutes "biochemistry" and particularly "medical biochemistry."

For the investigator the question of the content of his field is neither pertinent nor especially interesting. He is primarily concerned with research, the accomplishment of which depends upon the unity and continuity of knowledge, rather than upon its division. For the teacher, however, and more especially for the student, it is both convenient and important to know with some precision what body of knowledge is included in a given field of learning. This is especially true in medical biochemistry, for here much which will be important to the health and happiness of many, must be quickly and efficiently imparted to students who are already under heavy scholastic pressure. It is, therefore, of the very greatest importance to know what medical biochemistry is in order that it can be readily taught and as readily learned.

If science is the process of ascertaining the order of events in nature by repeated observations under similar conditions with a view to predicting the future, as Mast[1] maintains, then biochemistry can be defined as the process of ascertaining the order of chemical events in living organisms by repeated observations under conditions similar to those prevailing in the organism with a view to predicting these events. It follows from this that the biochemist utilizes chemical knowledge only in his methods and observation; his conclusions, his theories, and his philosophy are biological both in viewpoint and significance.

For the teacher of biochemistry in a medical college it is not enough alone to define biochemistry; the subject is too extensive. It includes general and comparative biochemistry, the biochemistry of the various divisions of the plant and animal kingdoms, and the biochemistry of various vital processes, organ systems, and cellular functions. In short, there is a biochemistry for every division and aspect of vital activity, not to mention the pedagogical partitions of the subject, one of which is medical biochemistry. The task of choosing from this vast quantity of knowledge the material which is pertinent for the medical student is no easy one.

If, however, the ends for which biochemistry is taught in the medical colleges are considered, the selection of the material to be included in the course will not be so difficult. These aims are as follows:

1. To provide an explanation of physiological phenomena by revealing the fundamental chemical events upon which they rest.

2. To provide a foundation in chemistry for instruction in related fields; for example, immunology, chemical therapeutics, pathological chemistry, dietetics, etc.

3. To provide an understanding of certain clinically important chemical tests and of the reasons for their importance.

A course designed to achieve these aims would probably have a content somewhat similar to the following:

I. Introduction to Biochemistry
   - A. Definition and Scope
   - B. Brief Historical Survey

II. The Physical Basis of Biochemistry
   - A. The Kinetic Theory
   - B. Phenomena of Polyphasic Systems
   - C. Phenomena of Surfaces

III. The Chemical Basis of Biochemistry
   - A. Some Chemical Equilibria
   - B. Equilibria of Biological Importance
   - C. Catalysis
   - D. Some Photochemical Effects of Biological Importance
   - E. The Chemical Constituents of Living Matter

IV. Metabolism
   - A. Correlation of Chemicophysical Principles with Vital Processes — Theories Concerning the Origin of the Metabolic Process
   - B. Metabolic Processes in General
   - C. The Metabolism of the Proteins*
   - D. The Metabolism of the Carbohydrates
   - E. The Metabolism of the Lipids and Related Substances
   - F. The Metabolism of Inorganic Substances
   - G. The Regulation of Metabolism

The second and third sections of the outline deal with the physical and chemical facts which serve as a basis upon which a discussion of the chemical events underlying mammalian, and particularly human, physiology must rest. The emphasis upon these sections will vary from year to year depending on the average background of the class. A detailed discussion of some point will be necessary for one class, while for another a brief reference will suffice. While it is the practice of many instructors to introduce and explain a chemical or physical fact when it is needed to elucidate a certain biochemical phenomenon, it would seem preferable to separate and systematize these facts for discussion by themselves. In this way they can be amplified or reviewed only briefly as the needs of the class indicate. This method also permits continual review and decreases the probability of incidental teaching which must be firmly avoided. The systematization of these facts and their separate discussion also gives to the course a very desirable continuity.

In the teaching of the physical and chemical bases of biochemistry to the medical student, it is necessary to remember that he is in the course of professional preparation and so the illustrations and examples presented for each point should be drawn, in so far as it is possible, from the field to which the student will later address himself.

Instruction in the chemical basis of biochemistry is given primarily to provide a foundation for the chemistry of metabolism. It is also designed to furnish a background for those other courses in the curriculum the thorough mastery of which depends upon a sound knowledge of the basic chemical phenomena. In this connection it must be noted that the teaching of the background for a course does not necessitate the teaching of the course itself. In short, the dividing line between medical biochemistry and pathological chemistry, immunology, chemical therapeutics, etc., while no doubt artificial, must be firmly preserved, if only for pedagogical reasons.

The part of the course concerning general metabolism provides a chemical basis for the comprehension of the overlying physiological phenomena. It is in this part of the course that so much of both the teaching and the textual material are confused. A large part of this confusion occurs because no clear distinction is made between the material which properly belongs to medical biochemistry and that which belongs to other subjects in the curriculum.

One of the principal difficulties is the uncertainty concerning what is characteristically biochemical and what physiological. A guide here is the fact that biochemistry must be confined to the fundamental chemical events underlying physiological activity. It is admittedly difficult to make this distinction, for a discussion of the biochemical events grades so imperceptibly into the overlying physiology that the latter is being taught before either the teacher or the students are aware of it. This is one of the hazards peculiar to the teaching of medical biochemistry and the instructor can not depend upon the standard texts for the removal of this handicap, for they themselves usually fail to make any clear distinction between the two subjects. Nevertheless, the difference must be realized both for economy of time and efficiency of instruction. Hence, the progressive instructor must, whether or no, prepare his lectures without benefit of a basic text, his material coming now from one text, now from another. The handicap, however, is not all upon the instructor, for the student in biochemistry, instead of comfortably supplementing his lecture notes from a basic text, must go to the library, there to use a different text for each part of the course, sometimes even two or three texts for the same topic.

For these reasons the instructor, in the preparation of his lectures, must be on his guard against wandering too far into physiology. The question will probably arise as to whether it makes any difference whether some physiology is taught in biochemistry or not. It is probably of no importance whether the material is taught in biochemistry or physiology as long as it is taught, but there is so much material properly biochemical that it would seem somewhat unwise, not to mention its being confusing to the student, to go into some other subject for material. Perhaps it would be a good plan if the departments of biochemistry

*The traditional divisions of metabolism are used here, although it is thought that its division into operative and energy-producing as given by Peters[3] would be somewhat more appropriate.

and physiology could arrange between them which was to teach the debatable topics. Thus much time would be saved through the avoidance of duplication and there would be more certainty in both departments that the ground has been adequately covered.

Parenthetically, it would probably be a good idea for all the departments in a medical college to compare their courses of study periodically. This would prevent duplication in the basic sciences and the clinical instructors would know better what to expect of the students in the way of preparation. By this means likewise the lack of coordination which is such a conspicuous feature of advanced education would in some measure be avoided.

Another of the causes of confusion in medical biochemistry is the obscurity concerning the relation between this subject which deals with the chemical events of the normal organism, and pathological chemistry which deals with the modification of these events by disease. Biochemistry is a preparation for the study of the chemistry of pathological conditions; it itself does not include pathological chemistry, although in the laboratory work of the course in medical biochemistry the two subjects ought to be closely related. A guiding principle in the teaching of pathological chemistry is the fact that pathological conditions are clinical entities and it would therefore seem best to take up the significant chemical events attendant upon a given pathology when that particular pathological condition is discussed by the instructor whose subject it is. Failing this preferred method, a separate course in pathological chemistry is indicated. Since changes in the chemistry of the body are usually of significance only in the degenerative diseases, this course could be short.

Another source of difficulty for the student is the discussion of the data and experiments of general biochemistry in too much detail. The discussions in the texts of the biochemistry of various animal forms are frequently carried to such lengths, because their authors intend the texts for use at both the professional and collegiate levels. A text written with this in view is usually not suitable, in its entirety at least, at either level. Teachers who diverge widely into general biochemistry usually make the excuse, and really believe, that the material is interesting, stimulating, and instructive. There can be little doubt that it is, but there is time only for so much material and a sufficiency of this can be found in medical biochemistry which is equally interesting, stimulating, instructive and, what is more, pertinent. It can not be emphasized too strongly, then, that medical biochemistry is concerned primarily with the human being and that the biochemistry of animal forms is relevant only when its implications are necessary for an understanding of the biochemistry of human beings.

Attention should also be called to the fact that medical students are professional students and not graduate students. For the latter, it is often necessary to know the details, even the minutiae, of an experiment; for the former, it is usually quite sufficient to know the results and the medical implications of an experiment.

The lecture method is under attack from so many quarters that some comment concerning this method of teaching would probably not be out of place. One of the most frequent statements of many modern educators is to the effect that lecturing is not teaching. If teaching is an endeavor on the part of the teacher whereby certain intellectual concepts, emotional attitudes, etc., are imparted to the student with a view to modifying his behavior, then lecturing is just as legitimate as any other method of accomplishing these ends. Of course, the lecture method as well as any other method is teaching only in so far as it changes the outlook and behavior of the student. The lecture method has several evident disadvantages, but so have all the other methods. Shaner[3] says concerning the lecture method: "But for technical and professional students the case is otherwise. Life is too short, the amount of exact information needed is too great, and the significance of much of it is too remote. A skillful lecturer is needed to guide the student through the maze of detail, to put first facts first, and to supply a temporary sustaining interest, that the student may hold his knowledge until experience provides a permanent interest. Much must be learned by rote. Why apologize for rote learning? Man and animals learn the fundamentals of daily living by rote, and understand why things are done so afterwards. It is the only practical way to learn many things."

A series of lectures based on an adequate amplification of the outline given above ought to provide a knowledge of the chemical events underlying physiological phenomena and a sufficient background for the later and more advanced courses in the professional curriculum. A series of even the very best lectures will not, however, provide the student with any skill in the performance of the clinically important chemical tests and without skill there can be little actual understanding of their significance. Such skill and understanding can be obtained only by adequate laboratory practice.

An examination of many laboratory manuals in biochemistry prepared for professional students shows that the experiments are seemingly chosen with hardly a thought to the future work of the physician. Some of these experiments are quite appropriate for the course in general biochemistry at the collegiate level: for example, The Interconversion of Aldoses and Ketoses, The Salting-Out Effect, The Preparation of Arginine, Influence of pH on the Swelling of Colloids, etc. Other experiments in these manuals are taken from quantitative and physical chemistry. Their purpose seems to be the instruction of the professional student in volumetric and gravimetric procedures, methods of calibration, etc. It is certainly true that many of the students coming to professional school are not acquainted with these technics, many of which would be very useful, but it is equally true that instruction in these technics is definitely the work of the instructors in collegiate chemistry and it is doubtful whether shortcomings in them can be remedied at the professional level.

In view of the experiments frequently presented in the laboratory of medical biochemistry, it is not surprising that so many physicians make little use of the diagnostic aids which biochemistry

offers them. They themselves have had too little experience with the clinically important chemical tests to understand their use. It is, therefore, of the utmost importance that what the student does in the biochemical laboratory will be useful in his later practice. Not that he will later perform most of those tests himself, but rather that he will understand them and thus know when and how they should be done and the meaning of their results.

A section near the end of most laboratory manuals is devoted to the clinical tests. This section ought to constitute the whole laboratory course. This, of course, would be possible only through the repetition of many experiments and the addition of others from various sources. A criterion for the selection of an experiment to be performed in the laboratory might well be a question concerning the clinical importance of the test to be chosen.

In the traditional method of laboratory instruction each student is permanently assigned to his place in the laboratory and has issued to him his own apparatus. Every student does the same series of experiments in nearly the same sequence and almost simultaneously. A teaching situation more nearly resembling the clinical laboratory results if each experiment with its accompanying apparatus and reagents is permanently arranged at a designated place in the laboratory. At the beginning of the period a student finds his assignment on a schedule prepared at the beginning of the semester. He then goes to the place in the laboratory where apparatus and reagents for the experiment or series of experiments are permanently set up and proceeds to work. His tests are performed on a sample which was put on the desk before the period began. This sample is prepared beforehand and contains the substance for which the examination is being made in either physiologically or pathologically probable concentrations. Its preparation must be very carefully done, for the student makes a report at the end of the laboratory period stating what concentration was present in the sample. If his report is not correct, he is required to repeat the experiment; this modification of the usual laboratory method has several advantages. The situation in the laboratory contributes to the reality of the work by duplicating to some extent the well-regulated clinical laboratory. There is probably a gain in the self-reliance of the student, for he must depend on himself, rather than on his neighbors, for they are not doing the same experiment. The student learns to perform the clinical tests more accurately than he otherwise would, for there is a premium on the carefulness of his work. Besides these pedagogical advantages, this method is economical, for it requires the use of comparatively few sets of apparatus. It has been found most efficient to have one assistant attend to the preparation of the samples and the verification of the reports; to have another prepare reagents and keep the stocks in order; and a third take care of the apparatus and keep account of the breakage of the apparatus by checking for each experiment immediately before and after each laboratory period. All the assistants, of course, aid in the laboratory in other ways. The system outlined has been used in the Kirksville College and has been found to be very satisfactory.

In preparing his lectures and the experiments for the laboratory it is important for the teacher of medical biochemistry to keep in mind precisely why his course is included in the professional curriculum and in teaching the course he must always be mindful of the intention of his students. He need not make them specialists; it is sufficient if he prepare them to be good physicians.

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Department of Professional Affairs

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Chairman
Cleveland

COMMITTEE ON SPECIAL MEMBERSHIP EFFORT

STEPHEN B. GIBBS, D.O.
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Coral Gables, Florida

EVERY MEMBER PAY HIS DUES AND GET A MEMBER!

At the time this is written, there are still too many who have not paid their annual A.O.A. dues. It would be regrettable should any of these be dropped through nonpayment. The Membership Committee has worked hard and made substantial gains. Are these gains to be offset through nonpayment of dues?

The A.O.A. is fighting a winning battle for our cause. There is no need to enumerate here the various accomplishments thus far. A sustained victory, however, will require the support of every osteopathic physician in our profession. That means A.O.A. membership. Our enemies have organization. Every member pay his dues and get a member.

S. B. G.

MEMBERSHIP REPORT AS OF DECEMBER 1, 1944

Membership count November 1, 1944		7,294
Applications received in November, 1944	22	
Graduates Licensed	28	
Restored to membership roll	2	
	52	
Less: Deaths and Resignations	11	
Net gain in November		41
Total membership count December 1, 1944		7,335

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